THE
LARYNGOSCOPE.

VOL. LXX MARCH, 1960 No. 3

A STUDY OF SPASTIC DYSPHONIA.

Neurologic and Electroencephalographic Abnormalities.*

EVELYN ROBE, Ph.D.,
JOEL BRUMLIK, M.D.,
and
PAUL MOORE, Ph.D.,

Chicago, Ill.

I. INTRODUCTION.

Spastic dysphonia, often referred to as hysterical dysphonia, has been considered a nosologic entity. It is usually not defined in terms of fundamental disturbances in neurophysiology. The study reported here, however, indicates that spastic dysphonia is a manifestation of disordered function of the central nervous system, and not a disease *in sui generis*.

Berendes[7,8] has described this condition as a disorder of phonation "which is characterized by a strained, creaking and choked vocal attack, tense and squeezed voice sound and is accompanied by extreme tension of the entire phonatory system." The present authors have also observed that there is usually a momentary phonatory interruption that occurs must frequently on initial vowel sounds. Whispered speech and extremely breathy phonation are ordinarily spoken without any hesitancy or other abnormal speech characteristics.

The present study was prompted, in part, by the singular

*From the Departments of Otolaryngology and Neurology and Psychiatry of Northwestern University Medical School, aided by a grant from the United Cerebral Palsy Research and Education Foundation (of Greater Chicago).

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Feb. 15, 1960.

lack of efficacy of voice therapy and psychotherapy in spastic dysphonia, and in part by the lack of constant anamnestic data. We have elected to study a group of patients with this clinical disorder for possible neurologic and electroencephalographic abnormalities. To our knowledge, there has been no previous attempt to define this disorder by parameters other than psychiatric.

Arnold[3] has suggested that spastic dysphonia might be due to removal of higher suppressor influences, allowing primitive subcortical centers to act without inhibition ("by the unconscious withdrawal of a shattered personality from the threats of daily life") ; but the fundamental neurophysiologic mechanism must be defined and the terminology made specific. The use of such a term as "suppressor influences" may refer to psychoanalytic suppression or to the function of one or more cortical suppressor strips. If a disease has a neurologic basis, the description of the underlying processes must be in specific terms germane to the field.

Segre[43,44] has stated that spastic dysphonia is an illness of the third and fourth decades of life. The ratio of males to females with this disease varies among authors. The precipating factors mentioned in the literature include excessive use of the voice and traumas of daily living. The authors beg to be excused from a discussion of psychodynamic abnormalities in spastic dysphonia for, while they may be present, this study is concerned specifically with the physiology of the central nervous system.

II. MATERIALS AND METHODS.

Ten patients with a clinical diagnosis of spastic dysphonia were selected for study. Each was given the five different types of tests and examinations indicated in the following paragraphs.

A. Examination of the Speech Organs and Adjacent Structures.

Each patient received a complete ear, nose and throat examination by at least two qualified otolaryngologists. This

evaluation included indirect laryngoscopy with special reference to a study of the movement of the true and false vocal cords. In addition, the nasal, pharyngeal and auditory passages were scrutinized. Three types of voice production were recorded for study: a standard passage was read; the total pitch range was sung; and a representative sample of conversational speech was spoken. Finally, a pure-tone audiometric examination was made for each subject, and additional special audiometric tests for central disturbances were administered to five of the patients.[21]

B. History.

An extensive history was obtained from each patient, including neurologic history with special reference to precipitating factors, familial neurologic disease and previous neurologic disease in the patient. A review of systems included specific questions as to a history of vertigo, diplopia, headaches, scotomata, weakness, paresthesias, unsteady gait, syncope and convulsions.

A history of the origin and development of the voice disturbance was obtained from each patient.

C. Neurologic Examination.

Each patient was given a complete neurologic examination, including an evaluation of the function of the cranial nerves, motor power, reflexes, coordination and sensation. A search was made not only for gross signs, but also each patient was combed for delicate signs of central nervous system disturbance.

D. Electroencephalographic Study.

Each patient was studied with standard electrode placement in frontal, anterior temporal, temporal, parietal and occipital areas. Monopolar and bipolar recording was used. Sleep tracings were obtained in all cases by the use of secobarbital sedation. Hyperventilation and photic drive stimulation techniques were employed. The alpha blocking response was recorded in all cases.

E. Laboratory Studies.

Those patients who merited further work-up received appropriate tests, including lumbar puncture, spinal fluid examination, skull and chest films, visual field examination, blood chemistries and urinalysis. Special tests, such as protein bound iodine and urine copper determinations, were performed as indicated.

TABLE I.

Examination of the Speech Apparatus and Adjacent Structures in Spastic Dysphonia.*

Patient	Age	Sex	Laryngoscopy	ENT Exam.	Audiometry	Recording†
S.S.	25	F	—	—	—	4
C.Y.	36	F	—	—	—	1
E.F.	38	F	—	—	—	1
E.T.	44	M	—	—	—	4
I.R.	45	F	—	—	—	4
S.K.	46	M	—	+	—	3
F.O.	50	F	—	—	—	3
J.O.	56	F	—	—	—	2
E.R.	59	F	—	—	—	2
M.C.	63	F	—	—	—	2-3

*See text for explanation. + = abnormal findings. — = negative.
†Scale of severity (average of the evaluation of four examiners): 1. mild; 2. moderate; 3. severe; 4. very severe.

III. RESULTS OF THE STUDY.

A. Examination of the Speech Apparatus and Adjacent Structures (See Table I).

The age range included in this study is 25 to 63 years. The male-female sex ratio is one to four. Laryngoscopy in all patients revealed normal structure. No sign of ventricular phonation was observed in any patient. Examination of the nasal, pharyngeal and auditory passages was completely normal in all cases except S. K., who had an anterior septal perforation.

Audiometry (pure-tone audiometry and special tests) was within normal limits in all cases. Recordings of voice were analyzed objectively and graded by four specialists in voice disorders according to the scale given in Table I.

TABLE II. HISTORICAL DATA IN SPASTIC DYSPHONIA.

Patient	Age Onset	Age Pt.	Duration (yrs.)	Hnd.[1]	Ppt.[2]	Fam.[3]	Neur.[4]	Ang.[5]	Nec.[6]	Fat.[7]	Prev. Rx[8]
S.S.	20	25	5	L	+	—	+	+	+	+	V, P
C.Y.	32	36	4	M	—	+	+	+	+	+	V
E.F.	24	38	14	R	—	+	+	+	+	+	—
E.T.	42	44	2	R	+	+	+	+	+	+	V, H
I.R.	30	45	15	R	+	+	+	+	+	+	V, P, A
S.K.	40	46	6	R	—	—	+	+	+	+	V
F.O.	47	50	3	R	+	—	—	—	+	+	V
J.O.	51	56	5	M	—	—	—	+	+	+	V
E.R.	56	59	3	R	+	—	+	+	+	+	V
M.C.	63	63	½	R	—	—	+	—	+	+	V
Per Cent					50	40	80	80	100	100	

[1]Handedness: L—Left; R—Right; M—Mixed.
[2]Precipitating Factors: A history of "cold," laryngitis or "flu" prior to the onset of voice disturbance.
[3]A family history of neurologic disease.
[4]A history of neurologic symptomatology in the patient.
[5]Anger: improvement in the voice problem when angry.
[6]Necessary: worsening of voice problem when speech is necessary.
[7]Fatigue: increase in voice disturbance with fatigue. [8]Previous Therapy: V—voice therapy; P—psychotherapy; H—hypnosis; A—intravenous amobarbital.

B. History (See Table II).

The duration of the illness ranges from six months to 15 years and apparently is not related to the patient's age at the time of onset. Seventy per cent of patients were right handed, 20 per cent ambidextrous and 10 per cent were left handed. Fifty per cent of patients had a history of "flu," "cold," "laryngitis" or upper respiratory infection immediately prior to the onset of voice disturbance. The remainder of patients either could not recall any precipitating factors or mentioned hard work, emotional trauma, etc., as associated with the onset.

Forty per cent of patients had a positive history of neurologic disease in the family. These statements included: *1.* "My mother was born blind because the optic nerves didn't develop" (C. Y.); *2.* a history of intention tremor of the head and hands in mother, sister and brother (E. F.); *3.* a seizure disorder in the father of the patient (E.T.); and *4.* "My mother had a stroke" (I. R.).

Eighty per cent of the patients had a positive history of neurologic disturbance prior to the onset of voice difficulty. These symptoms included: *1.* transient scotomata in both eyes and transient weakness of both legs (S. S.); *2.* tremor of the head and hands of the intention type (E.F.); *3.* paralysis of the lower extremities as a child (S. K.); *4.* a transient episode of clumsiness and weakness of the left leg accompanied by dizziness (E. R.); *5.* several episodes of syncope without convulsions (C. Y.); *6.* attacks of syncope especially associated with coughing and periods of confusion with transient paresthesias in one thumb (E. T.); *7.* paresthesias noted on occasion in the left hand (I. R.); and *8.* intermittent dysphagia (M. C.).

Eighty per cent gave a history of improvement in their voices with emotion (anger). One hundred per cent stated that situations in which talking was necessary aggravated their voice disturbance. Fatigue was universally effective in worsening the symptoms; otherwise, historical findings were not contributory.

TABLE III.

Clinical Findings in Spastic Dysphonia.

S. S.
1. Bitemporal pallor of the optic discs.
2. Absent abdominal reflexes.
3. The left angle of the mouth pulls away better than the right and the patient talks better out of the left side of her face; however, this appears to be an asymmetry rather than a true neurological lesion.
4. Questionable left pronation sign.
5. Hearing lateralizes slightly to the left.
6. A tendency to become unsteady as she walks in tandem.
7. A suggestion of an adductor response bilaterally when the knee jerks are elicited.

C. Y.
1. Minimal fine rotary tremor of the head of the intention type.
2. Absent abdominal reflexes.

E. F.
1. Intention tremor, involving the head and both upper extremities, perhaps more so on the left.
2. Definite ataxia as the patient walks in tandem, tending to fall to the left.
3. Deep tendon reflexes of the lower extremities are slightly more brisk on the left with an adductor response on both sides as the knee jerks are elicited, perhaps more prominent on the left.
4. The abdominal reflexes are absent.
5. There is minimal falling away on both sides as the opposite arm past points.
6. The patient tends to swing her right arm better than her left as she walks.

E. T.
1. The tongue protrudes to the left.
2. There is minimal falling away of the right upper extremity.
3. The right lower limb lies slightly externally rotated as the patient lies upon the examining table.
4. The patient swings his left arm slightly better than his right as he walks.
5. The left abdominals are greater than the right and the left cremasteric is greater than the right.
6. The right knee jerk is slightly greater than the left.

I. R.
1. The left pupil is slightly larger than the right.
2. The left face is weak in the manner of a supranuclear facial paresis.
3. The left biceps, radial and ulnar jerks are greater than the right.
4. The right knee and ankle jerks are greater than the left.
5. The right lower limb lies slightly externally rotated on the examining table.
6. There are questionable Trömner, Gonda and Oppenheim responses on the left.
7. There is unsustained nystagmus on right lateral gaze with the slow component to the left.

S. K.
1. The left angle of the mouth pulls away better than the right.
2. The left platysma contracts better than the right.
3. The patient smiles and talks better out of the left side of the face.
4. Minimal right pronation sign and falling away.
5. The left lower limb lies externally rotated.
6. The left biceps, triceps, radial, knee and ankle jerks are greater than the right.

TABLE III (Continued).

7. The left abdominals and cremasterics are greater than the right.
8. Minimal intention tremor bilaterally.
9. Positive Quinquaud sign.
10. The right arm swings less well than the left as the patient walks.

F. O.

1. The left pupil is slightly greater than the right.
2. The tongue protrudes slightly to the right.
3. The right side of the face pulls away a little better than the left. The right platysma contracts slightly better than the left. The patient talks a little better out of the right side of her face.
4. The left biceps, radial and ulnar and knee jerks are slightly greater than the right.
5. The patient swings her right arm a little better than the left as she walks.
6. The abdominal reflexes are absent.

J. O.

1. The left pupil is slightly larger than the right.
2. The right ankle jerk is slightly greater than the left.
3. There is an adductor response on eliciting the knee jerks on both sides.
4. The right abdominals are slightly more brisk than the left.
5. The patient swings her left arm slightly better than the right as she walks.
6. There is a more definite flexor response on stroking the right planta than the left.
7. There is a fine tremor of the closed eyelids.

E. R.

1. Definite snout reflex.
2. The left radial reflex is greater than the right.
3. The right knee jerk is greater than the left.
4. There is a definite left Gonda.
5. A minimal falling away of both extremities as the opposite arm past points.
6. There is a slight left pronation sign.
7. The patient tends to swing her right arm less well than the left as she walks.
8. The left lower limb lies slightly externally rotated as the patient lies upon the examining table.
9. The patient tends to become unsteady as she walks in tandem and with standing on either foot alone, especially on the left.

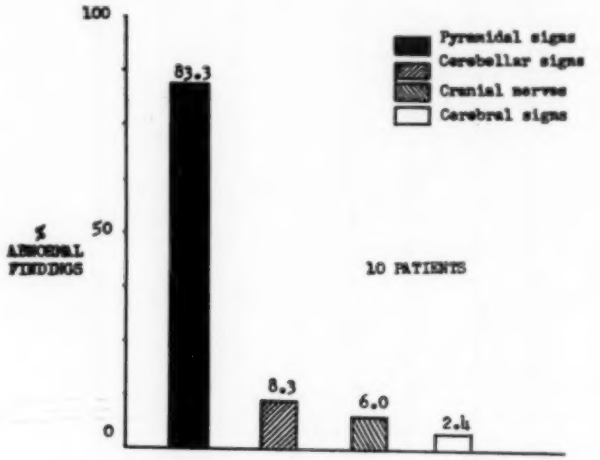
M. C.

1. A positive snout reflex.
2. The right angle of the mouth pulls away slightly better than the left and the patient talks better out of the right side of her face.
3. There are bilateral Babinski hand signs, more marked on the right.
4. The abdominal reflexes are not elicited.
5. The right ankle jerk is greater than the left.
6. There is a right Gordon.
7. There are bilateral Hoffmann signs and Trömners.

Ninety per cent of patients had some previous therapy for their voices. Ninety per cent had voice therapy from one or more therapists, 20 per cent had psychotherapy, and one patient each had hypnosis and intravenous amobarbital. All these therapies were strikingly ineffective.

C. Neurologic Examination (See Table III and Figs. 1 and 4).

All patients had neurologic signs indicating disease of the central nervous system. A total of 84 neurologic signs was elicited, approximately eight per patient. By far the most frequently observed abnormalities were pyramidal tract signs (83 per cent), with cerebellar signs also relatively frequent (8 per cent).



NATURE OF ABNORMAL NEUROLOGICAL SIGNS IN SPASTIC DYSPHONIA

(Total abnormal signs = 84 = 8.4 signs per patient)

Fig. 1.

Forty-three per cent of signs lateralized to the right and 32 per cent to the left cerebral hemisphere (25 per cent were bilateral). If bilateral signs are excluded, right cerebral signs increase to 57 per cent and the left-sided signs to 43 per cent.

Coupled with the medical and neurological history, an impression of specific neurologic disease was made in four patients: *1.* heredofamilial tremor (E.R.); *2.* small cerebral

REPRESENTATIVE ELECTROENCEPHALOGRAMS.

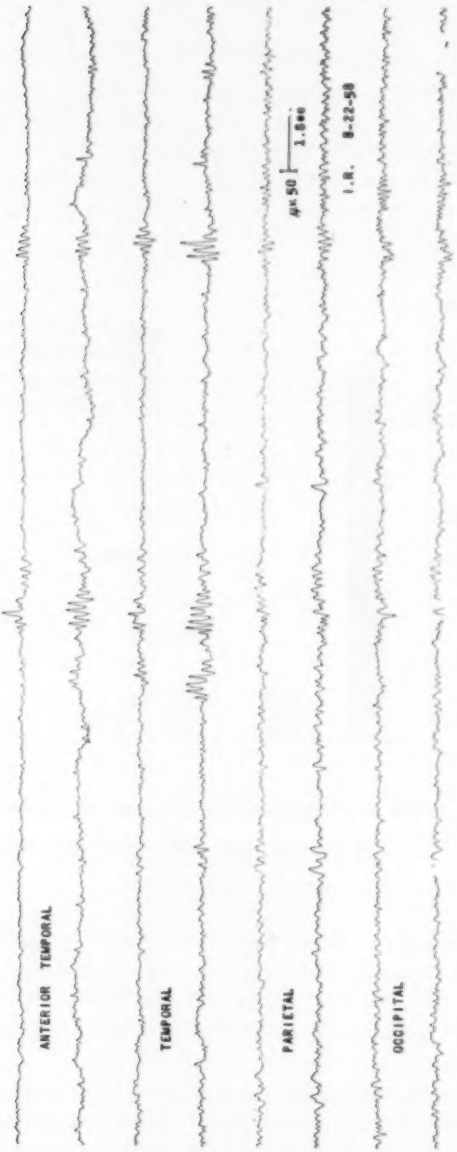


Fig. 2-a.

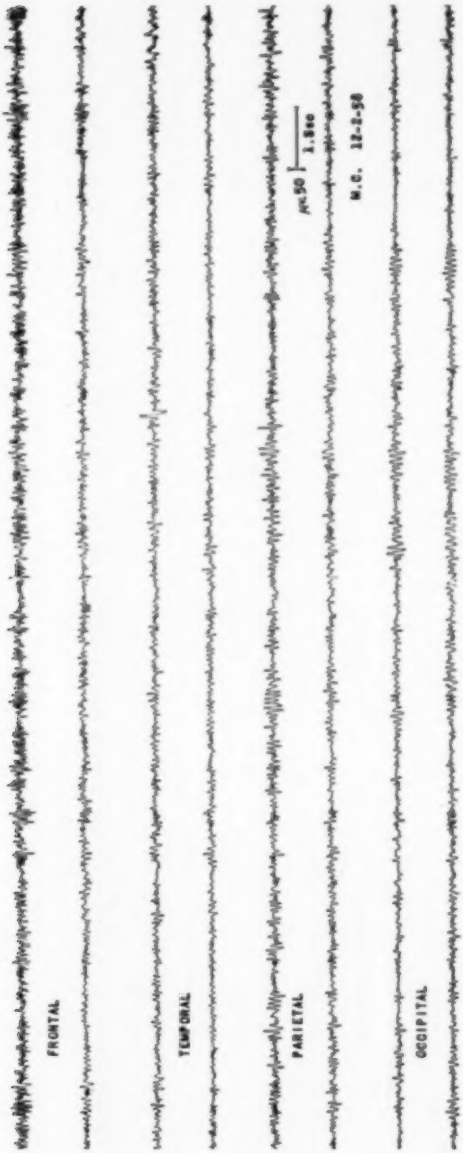


FIG. 2-b.

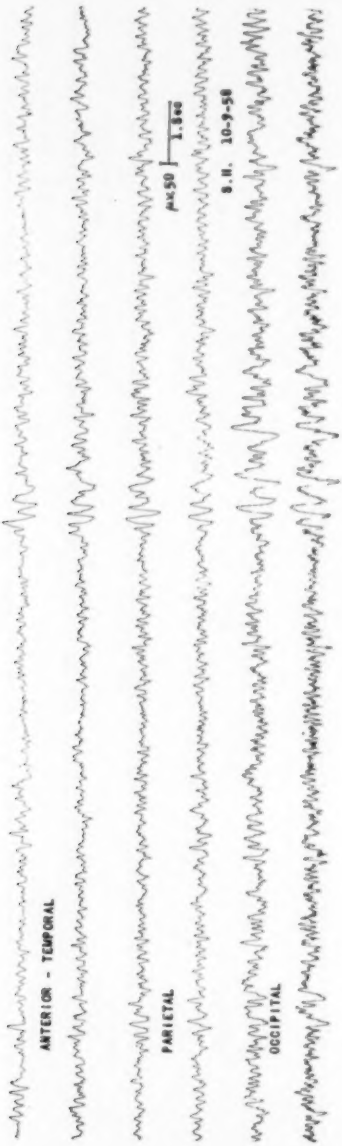


FIG. 2-c.

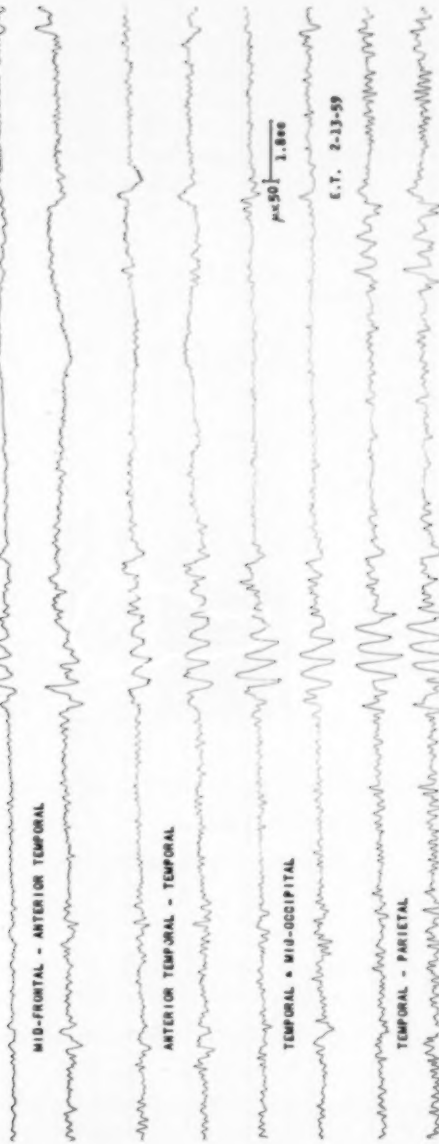

MID-FRONTAL - ANTERIOR TEMPORAL
ANTERIOR TEMPORAL - TEMPORAL
TEMPORAL - MID-OCCIPITAL
E.T. 2-13-59
TEMPORAL - PARIETAL

Fig. 2-d.

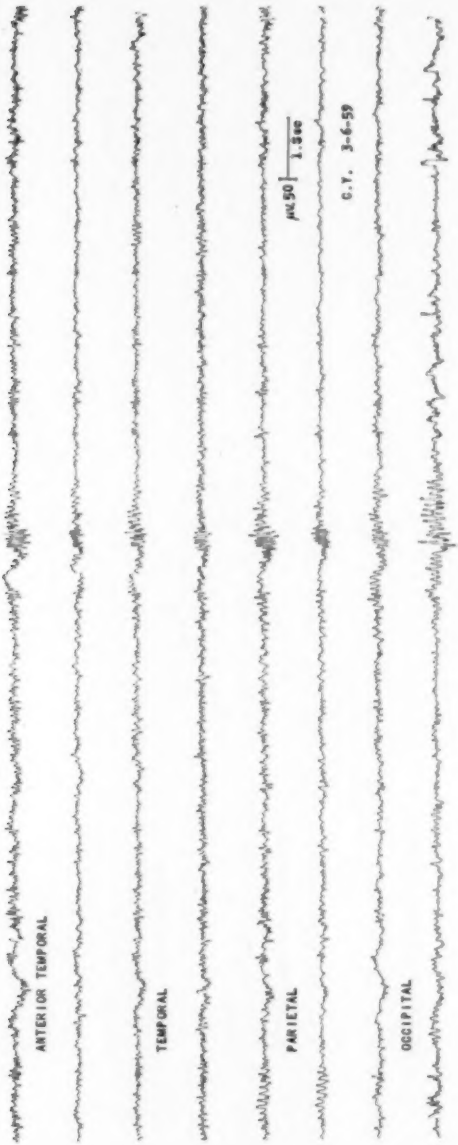
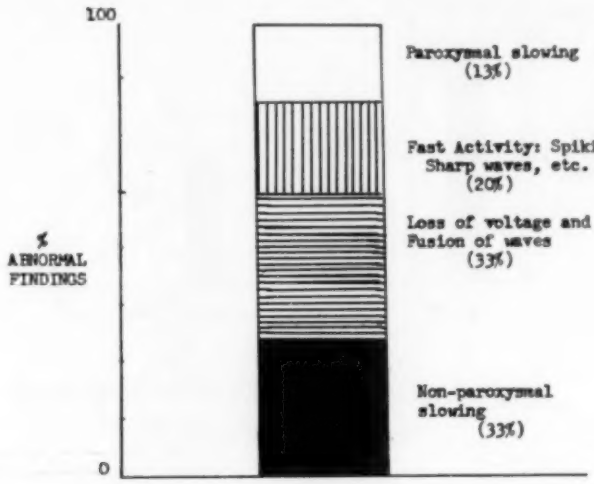

ANTERIOR TEMPORAL
TEMPORAL
PARIETAL
μv.50 / 1 sec
C.V. 3-6-59
OCCIPITAL

Fig. 2-c.

thromboses in the frontal area (arteriosclerosis) (M.C.); *3.* transient cerebral ischemic attacks (E.R.); and *4.* multiple sclerosis (S. S.). The remaining patients had signs and symptoms which did not point to distinct clinical entities, but rather to a diffuse or disseminated disease of the pyramidal and extrapyramidal systems. The etiology of the process



THE NATURE OF THE ELECTROENCEPHALOGRAPHIC ABNORMALITIES IN SPASTIC DYSPHONIA

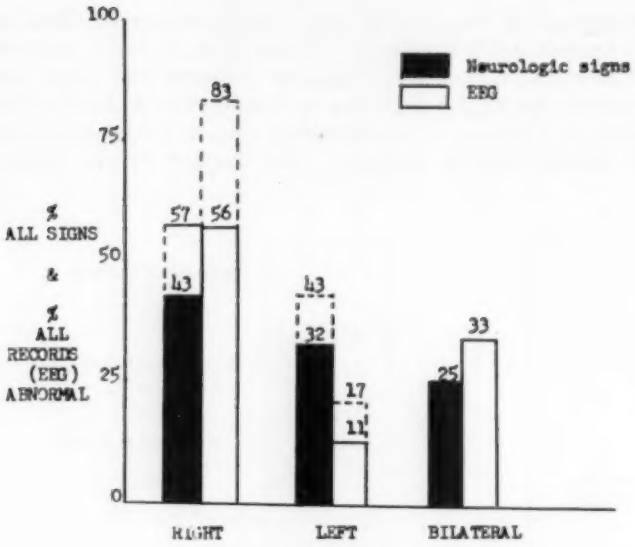
(10 PATIENTS. 9 ABNORMAL EEGs)

Fig. 3.

was not obvious in these cases. The possibility of a post-encephalitic process will be discussed below.

D. Electroencephalographic Disturbances (See Figs. 2, 3, 4 and 5).

Nine of the ten electroencephalograms taken were abnormal (E. F. was normal).



THE LATERALITY OF DISTURBANCE IN SPASTIC DISPHONIA:
THE NEUROLOGICAL EXAMINATION
COMPARED WITH THE ELECTROENCEPHALOGRAM.

(Dotted lines indicate the % lateralizing signs and records)

Fig. 4.

Fifty per cent of patients had records compatible with burst-like discharges.* Of these five patients, three had findings primarily parietal in location and two mainly frontal. In three cases, the findings were bilateral, in one case primarily right sided, and in one case mainly left sided. The nature of these paroxysmal discharges were high voltage fast activity in three cases and hypersynchronous slowing in two cases.

*Though two patients mentioned syncope, no patient had a history of clinical seizures.

In considering all abnormal findings in the nine cases of abnormal electroencephalograms, no predominant type of abnormality was observed. Burst-like discharges (high voltage fast activity and hypersynchronous paroxysmal slowing) were seen in 33 per cent of tracings. A reduction in voltage and basic frequency was observed in another 33 per cent of findings, predominantly in the right temporal and right

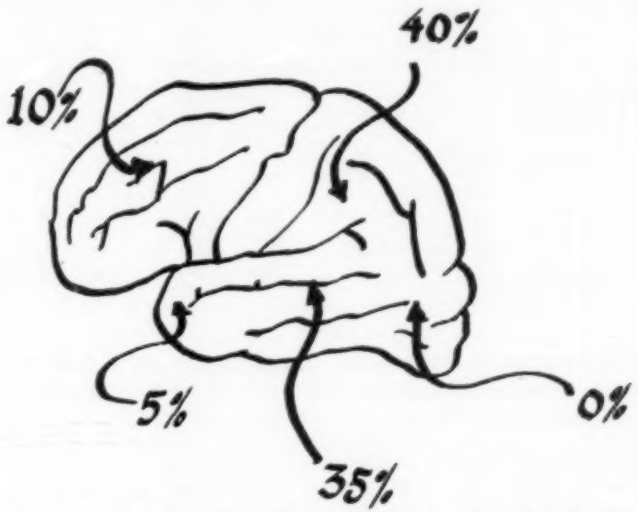


Fig. 5. Localization of all EEG findings in spastic dysphonia (10 patients).

parietal areas. Non-paroxysmal slowing constituted the remaining third of the disturbances.

The laterality of these abnormalities was 56 per cent right sided, 11 per cent left sided and 33 per cent bilateral. Excluding the latter, the right sided findings rise to 83 per cent and the left sided disturbances to 17 per cent.

The location of these findings (see Fig. 5) is 15 per cent for frontal, anterior temporal and occipital areas combined, as opposed to 75 per cent for temporal-parietal location.

In summary, the electroencephalographic findings suggest a cortical disturbance in the temporo-parietal area in the right cerebral hemisphere and paroxysmal in nature.

E. Laboratory Results (see Table IV).

In four cases out of five with burst-like EEG discharges, the patients were hospitalized for further work-up as indicated in Table IV. No spinal fluid finding was abnormal.

TABLE IV.

Laboratory Results in Spastic Dysphonia.

Patient	Visual Field Examination (Central)	Skull Films	Cerebrospinal Fluid Examination*				
			Wassermann	Protein (mg%) Tube 1	Protein (mg%) Tube 3	Cl	Gold curve
S.S.	Peripheral constriction (to red)	Small punched-out lesions (vascular lakes?)	Neg.	41.5	33.0	718	0111000000
C.Y.	Negative	Negative	Neg.	29.5	28.0	730	0011000000
I.R.	Negative	Osteoma, rt. occiptal bone	Neg.	34.5	34.5	717	0111110000
E.T.	Negative	Negative	Neg.	29.5	29.0	742	0111000000

*All cell counts within normal limits.

Skull films revealed only incidental pathology (S. S. and I. R.). Central visual field examinations revealed pathology (peripheral constriction) in one case (S. S.).

A high erythrocyte sedimentation rate was found in three patients (S. S.: 35; I. R.: 23; J. O.: 27) and glycosuria was noted in one patient (I. R.); otherwise, blood chemistries were within normal limits, including a protein bound iodine (S. K.) and a urine copper determination (E. F.).

IV. DISCUSSION.

A survey of the experimental neurophysiology of phonation and the contributions of clinical neurology are pertinent to the present discussion. Both fields of study offer evidence to explain the phenomena of spastic dysphonia.

A. The Neurophysiology of Vocalization.

1. The frontal-parietal areas: Furstenberg[16] has provided direct evidence that laryngeal closure may follow cortical

stimulation. The larynx of primates was visualized by direct laryngoscopy and various foci on the cortex were stimulated. Laryngeal closure, with either apnea or diaphragmatic contraction, resulted from stimulation of five areas: *1.* the anterior temporal lobe; *2.* the frontal operculum; *3.* the anterior island of Reil; *4.* the temporal-parietal area; and *5.* the ventral part of the motor cortex. The lowest threshold for response was associated with the first-named area. Sugar, et al.,[46] has described a second motor area in the monkey, buried deep in the Sylvian fissure. When the para-insular portion is stimulated, adduction-abduction movements of the vocal cords are observed under direct vision. From the adjacent insula, stimulation produced respiratory slowing and arrest.

Penfield and Jasper[38] describe vocalization that follows stimulation of the motor cortex (lower part of area 4) as a long-drawn-out sound made by symmetrical use of mouth, pharynx, larynx and muscles of mastication. Occasionally respiratory arrest has been produced by stimulation of the lower part of area 4. Penfield and Rasmussen[40] describe similar localization. Either speech arrest or vocalization may be produced by stimulation of *1.* the superior frontal gyrus, or *2.* the lower part of area 4. So-called aphasic arrest can be elicited by stimulating *1.* area 44, *2.* the supramarginal gyrus, and *3.* the posterior temporal lobe; moreover, removal of the pre-central gyrus will cause a temporary dysphagia and dysphonia. The authors believe that since these speech areas can be isolated from the rest of the cortex without producing disturbance in speech, the pathways for vocalization are subcortical.

2. The temporal lobe: Kaada[22] described various rhinencephalic areas in lower animals which produce alterations in respiration following stimulation. The extrapyramidal nature of the pathways involved was demonstrated. Other authors have confirmed the observation that stimulation of the temporal tip, limbic area and amygdaloid complex can produce respiratory arrest in lower animals[13]; moreover, from these areas (in the monkey),[45] vocal responses have been produced similar to those the animal usually makes. Characteristically, the sound continues after the stimulation stops.

Baldwin, et al.,[5] noted that a patient reported at surgery for temporal lobectomy, "I felt like my throat was closing up somewhat, and I could not talk." This comment was made as the para-amygdalar area was stimulated.

Ablation of the tip of the temporal lobe also has its effect on speech production. Bucy[10] states that, following temporal lobectomy in monkeys, there is a loss of vocal activity—the animals remain mute. Removal of the temporal lobes in man produces profound emotional changes. Boshes[9] has stressed the intimate relationship of the temporal lobe to the limbic system and emotion. Other effects of temporal lobectomy have been outlined by Bucy and Kluver.[11]

Thus the temporal lobe is intimately connected with emotional-behavioral responses on the one hand, and autonomic nervous system responses on the other, and is related to vocalization and respiration. The subcortical pathways needed for cortical expression have been outlined anatomically and physiologically[1,4,18,23,28,30,42,48] as they proceed through the extra pyramidal system. An interesting condition known as "pathological" or "involuntary" laughing and crying has led Monrad-Krohn[32] and Wilson[47] to postulate separate motor systems for "voluntary motion" and "emotional movement." Monrad-Krohn states that the latter is normally under suppressor control from the former and may appear in disease states. Wilson, in 1924,[47] postulated a coordinated facio-vocal-respiratory center in the upper pons, which is normally under double control—emotional and voluntary—with symptoms resulting if one or the other is destroyed. He cites the pathological laughing of the patient with pseudobulbar palsy as an example of destruction of the voluntary system, and the masked facies of the Parkinsonian as a loss of emotional system function.

3. The brainstem: That a center for vocalization exists in the brainstem has been demonstrated by Magoun, et al.[31] Stimulation of the periaqueductal gray matter and tegmentum in cats and monkeys produces coordinated facio-vocal activity resembling that seen during unpleasant emotion in the normal animal. Such responses are seen from stimulation of the hypothalamus and in the decerebrate cat; hence this must be

an "efferent" center. While stimulation proceeds, various sympathomimetic effects, including mydriasis, are noted. Luchsinger[29] reported mydriasis as a specific accompaniment of attempted speech in the patient with spastic dysphonia. In addition, ipsilateral mydriasis follows amygdaloid stimulation.[26] Kelly, et al.,[24] abolished facio-vocal activity by lesions placed in the periaqueductal gray matter and tegmentum of cats. The authors state that this is a specific center and is concerned not solely with the integration of facio-vocal impulses but also with the precipitation of their integrated activity "at times when it forms an appropriate part of the animal as a whole."

B. Contributions of Clinical Neurology.

Classical neurology has concerned itself largely with the effects of ablative cortical lesions on the speech mechanism. The cardinal sign of cortical speech dysfunction is an alteration of language. Hughlings Jackson[20] distinguishes two varieties of speech: automatic and intellectual. An aphasic patient may lose voluntary (intellectual or proportional) language, but retain emotional speech, *i.e.*, he may laugh, cry, vary the tone of his voice and sing. This is extremely reminiscent of the so-called "psychogenic" variations in the voice disturbance in spastic dysphonia. Hughlings Jackson felt that the right (non-dominant) cerebral hemisphere was for automatic, and the left hemisphere was for intellectual, speech. Kussmaul[27] mentions pseudobulbar palsy, where voluntary speech may be paralyzed but speech accompanying emotion is preserved; moreover, the symptoms of organic neurologic disease are frequently altered by emotion.[37]

As far as the role of the non-dominant hemisphere in speech is concerned, very little basic information is extant. Nielsen[34] went so far as to say: "Even today there is no unanimity of opinion concerning localization of cerebral function in the sphere of language, and this lack of agreement is dependent on our ignorance of the function of the minor cerebral hemisphere." Nielsen has little new to add on the subject[33,34,35] and cites the theory of Jackson as the probable function of the non-dominant hemisphere in speech. Both Head[19] and Freud[14]

agree that it is dangerous to define well-circumscribed speech areas in the cortex. The process of speech production is a dynamic one, involving countless reverberating circuits. These clinical observations on the role of the non-dominant hemisphere in the production of language disturbances correlate well with the lateralization of abnormalities in this study.

The literature on the temporal-parietal junction is also meager, being confined to ablative procedures on the dominant side. There is no doubt, however, that the angular and supramarginal gyri are essential to the circuitry of speech. Lesions of these areas produce the syndromes of word-blindness and word-deafness, and every clinician is familiar with the sensory aphasia resulting from such lesions. Irritative cortical disturbances in these areas are infrequently reported in the literature, and with even greater rarity on the minor side.[39,25,41,12] No evidence of abnormal phonation due to irritation of the cortical speech areas (with special reference to the temporal-parietal junction) can be found; however, cases of stuttering associated with temporal lobe epilepsy have been reported[2] in which there were focal findings on electroencephalograms of the right temporal area.

In summary, there is no evidence in the literature for a disorder of phonation accompanying a cortical disturbance in the area indicated by our study, although lesions in this area are known to produce aphasia, and the temporal-parietal junction is a recognized "speech area." It is possible that the disturbance in spastic dysphonia is partially subcortical and that the cortex is involved by electrical excitation along corticopetal pathways; however, the laterality of the disturbances and the fast activity suggest primary cortical abnormality. It is possible that both cortical and subcortical disturbances exist, the abnormal EEG pattern being the result of more than one lesion.

C. Discussion of Results of the Present Study.

The examiner received the impression of a specific neurologic entity in four of the ten cases. We do not attempt to ascribe undue significance to small neurologic signs nor do we wish to weigh the laterality percentages too heavily;

yet that this small group of patients manifests so many and such significant signs, leads to the admonition to perform a minute neurologic examination on each patient with spastic dysphonia. The patient with this disease almost assuredly will have neurologic abnormalities.

The authors believe that the most significant findings in this study are electroencephalographic. Several conclusions can be drawn: *1.* there is an electrical cortical disturbance in spastic dysphonia, though the primary disturbance may be partially subcortical and cause cortical disturbances over the projection systems; *2.* the commonest location of the disturbance is temporal-parietal; *3.* its laterality is primarily in the right (non-dominant) cerebral hemisphere; and *4.* its nature is mainly burst-like or paroxysmal. Of interest are electroencephalographic findings in stutterers which are almost identical to those found in this study.[36,6] Coupled with the neurologic findings in these patients, a disturbance in neural function and possibly structure is highly suspect, if not definite, in spastic dysphonia.

Several patients have noted difficulty in speaking when they *have* to, but ease up when they are not so pressed or when swearing. This is reminiscent not only of certain aphasic patients (see above) but also of the "separate" emotional and voluntary motor systems proposed by Monrad-Krohn and Wilson[32,47] Arnold[3] mentioned that whispering, choral speaking, singing, talking in sleep are not affected in spastic dysphonia. Coughing, laughing and moaning are preserved. "Even singing may be perfectly normal as long as the patient does not attach any meaning to his emotional expression . . ."[3]

We are impressed by the high incidence of extrapyramidal signs in our patients. This would point to disturbances in subcortical pathways, which every author seems to think are the paths for speech production; moreover, the intimate connection of the temporal lobe with the pathways for emotional expression suggest a possible mechanism by which psychic factors alter the symptoms of spastic dysphonia.

Finally a possible post-encephalitic etiology in some cases of spastic dysphonia is suggested by the incidence of upper

respiratory infections prior to the onset of voice disturbance. Such viral infections have a predilection for subcortical structures, produce EEG findings similar to those in this study, and may antedate the onset of neurologic symptoms by many years; however, the variability of symptoms, signs and course from patient to patient precludes a general statement.

The authors do not mean to imply that there are no psychiatric abnormalities in spastic dysphonia, or that psychotherapy does not have a place in treatment (as it has in the treatment of all chronic diseases); however, we wish to emphasize that each patient with spastic dysphonia should have a neurologic work-up which will more than likely be illuminating, and that spastic dysphonia has a neurologic basis, probably of specific location, but most likely of varied etiology. It cannot be stated that the neurologic and electroencephalographic abnormalities are related to the voice disturbance as cause and effect; yet such a strong association suggests that this phonatory disturbance is not a clinical entity, but a *symptom* in a broader syndrome of neurologic disease. Just as a fever prompts the physician to search for the underlying disease process, so should the symptoms of spastic dysphonia indicate an examination of the patient for disease of the central nervous system.

V. SUMMARY AND CONCLUSIONS.

1. Ten patients with spastic dysphonia were studied with neurologic examinations and electroencephalograms.

2. Every patient had signs of central nervous system disease, with suggestive specific clinical entities in four cases.

3. Ninety per cent of electroencephalograms were abnormal, with the disturbance generally being paroxysmal in the right temporal-parietal area.

4. The authors consider spastic dysphonia to be a symptom of disease of the central nervous system, both from the standpoint of this study and from the clinical and experimental facts in the literature.

5. It is proposed that there is a specific disturbance in the

pathways for speech production, in some cases perhaps on a post-encephalitic basis.

ACKNOWLEDGEMENT.

The authors express their appreciation to Dr. Ray S. Snider, Dr. Benjamin Boshes and Dr. Frank Schmid for their valuable assistance in this study.

BIBLIOGRAPHY.

1. Ajmone-Marsan, C., and Stoll, J.: Subcortical Connections of the Temporal Pole in Relation to Temporal Lobe Seizures. *Arch. Neurol. and Psych.*, 66:669-686, 1951.

2. Anastasopoulos, G.; Diacoyannis, Ag., and Routsonis, K.: Bégaiement et Épilepsie Temporale. *Rev. Neurol.*, 99:472-479, 1958.

3. Arnold, Godfrey: Spastic Dysphonia: 1. Changing Interpretations of a Persistent Affliction. *Logos*, 2:3, 1959.

4. Bailey, P.; Bonin, G. v.; Garol, H. W., and McCulloch, W. S.: Functional Organization of Temporal Lobe of Monkey (Macaca mulatta) and Chimpanzee (Pan satyrus). *Jour. Neurolphysiol.*, 6:121-128, 1943.

5. Baldwin, M.; Frost, L. L., and Wood, C. D.: Investigation of the Primate Amygdala: Movements of the Face and Jaws. *Neurology*, 4:586-598, 1954.

6. Bente, D.; Schönhärl, E., and Krump, J.: Elektroencephalographische Befunde bei Stottern und ihre Bedeutung für die medikamentöse Therapie. *Arch. f. Ohren-Nasen-und Kehlkopfheilk.*, 169:513-519, 1956.

7. Berendes, J.: Zur Entstehung und Behandling der Dysphonia Spastica. *Zeitschr. f. Hals u.s.w. Heilk.*, 44:78, 1938.

8. Berendes, J.: Spastische Dysphonie. *Arch.. Sprach - und Stimmheilk.*, 3:29, 86, 189, 1939.

9. Boshes, B.: Emotions, Hypothalamus and the Cardiovascular System. *Amer. Jour. Cardiol.*, 1:212-223, 1958.

10. Bucy, P. C.: Personal communication, 1959.

11. Bucy, P. C., and Kluver, H.: Anatomic Changes Secondary to Temporal Lobectomy. *Arch. Neurol. and Psych.*, 44:1142-1146, 1940.

12. Chavany, J. A.; Lobel, G., and Hagenmüller, D.: Des Troubles du Language d'Essence Epileptique. *Presse Med.*, 64:1449-1451, 1956.

13. Clark, G.; Chow, K. L.; Gillaspy, C. C., and Klotz, D. A.: Stimulation of the Anterior Limbic Region in Dogs. *Jour. Neurophysiol.*, 12:459-463, 1949.

14. Freud, S.: "On Aphasia," 105 pp. International Universities Press, New York, 1953.

15. Frost, L. L.; Baldwin, M., and Wood, C. D.: Investigation of the Primate Amygdala: Movements of the Face and Jaws. After-discharge and the Anterior Commissure. *Neurology*, 8:543-546, 1958.

16. Furstenberg, A. C.: Evidence of Laryngeal Participation in Emo-

tional Expression: Its Relation to Hysterical Aphonia. *Ann. Otol., Rhinol. and Laryngol.*, 67:516-527, 1958.

17. Gloor, P.: Electrophysiological Studies of the Amygdaloid Nucleus in the Cat. I. The Neuronal Organization of the Amygdaloid Projection System. *EEG Clin. Neurophysiol.*, 7:223-242, 1955.

18. Gloor, P.: Electrophysiological Studies on the Connections of the Amygdaloid Nucleus in the Cat. Part II: The Electrophysiological Properties of the Amygdaloid Projection System. *EEG Clin. Neurophysiol.*, 7:243-264, 1955.

19. Head, H.: Speech and Cerebral Localization.*Brain*, 46:355-528, 1923.

20. Head, H.: Hughlings Jackson on Aphasia and Kindred Affections of Speech. *Brain*, 38:1-190, 1915.

21. Jerger, J., and Harford, E. R.: The Alternate and Simultaneous Balancing of Pure Tones. *Jour. Speech and Hear. Res.*, in press.

22. Kaada, B. R.: Somato-motor, Autonomic and Electrocorticographic Responses to Electrical Stimulation of Rhinencephalic and Other Structures in Primates, Cat and Dog. *Acta Physiol. Scand.*, 23: suppl. 83, 1951.

23. Kaada, B. R.; Anderson, P., and Jansen, J., Jr.: Stimulation of the Amygdaloid Nuclear Complex in Unanesthetized Cats. *Neurology*, 4:48-64, 1954.

24. Kelly, A. H.; Beaton, L. E., and Magoun, H. W.: A Midbrain Mechanism for Facio-vocal Activity. *Jour. Neurophysiol.*, 9:181-189, 1946.

25. Kisker, K. P.: Sprachliche Stereotypen bei Temporallapen-Epilepsie. *Nervenarzt*, 28:366-368, 1957.

26. Koikegami, H., and Yoshida, K.: Pupillary Dilatation Induced by Stimulation of Amygdaloid Nuclei. *Fol. Psychiat. and Neurol., Japan*, 7:109-126, 1953.

27. Kussmaul, A.: "Die Störungen der Sprache." F. C. W. Vogel, Leipzig, 1877.

28. Lauer, E. W.: The Nuclear Pattern and Fiber Connections of Certain Basal Telencephalic Centers in the Macaque. *Jour. Comp. Neurol.*, 82:215-254, 1945.

29. Luchsinger, R.: Das Vegetative System bei Krämpfartigen Sprachstörungen. *Pract. Oto-Rhino-Laryngol.*, 6:204-213, 1944.

30. MacLean, P. D., and Delgado, J. M. R.: Electrical and Chemical Stimulation of Fronto-temporal Portion of Limbic System in the Waking Animal. *EEG and Clin. Neurophysiol.*, 5:91-100, 1953.

31. Magoun, H. W.; Atlas, D.; Ingersoll, E. H., and Ranson, S. W.: Associated Facial, Vocal, and Respiratory Components of Emotional Expression: an Experimental Study. *Jour. Neurol. and Psychopath.*, 17:241-255, 1937.

32. Monrad-Krohn, G. H.: On the Dissociation of Voluntary and Emotional Innervation in Facial Paresis of Central Origin. *Brain*, 47:22-35, 1924.

33. Nielsen, J. M.: Agnosias, Apraxias, Speech and Aphasia. "Clinical Neurology," Vol. I, Chap. 4 (A. B. Baker, Ed.). Paul B. Hoeber, New York, 1955.

34. Nielsen, J. M.: Function of the Minor (Usually Right) Cerebral Hemisphere in Language. *Bull. for Los Ang. Neurol. Soc.*, 3:67-75, 1938.

35. Nielsen, J. M.: "Agnosia, Apraxia, Aphasia: Their Value in Cerebral Localization," 292 pp. Paul B. Hoeber, Inc., New York, 1946.

36. Ottoboni, A., and Grossi, Bianchi, M. L.: Indagine Elettroencefalografica in Bambini Balbuzienti. *Min. Otorinolaring.*, 64:215-216, 1946.

37. Parker, H. L.: "Clinical Studies in Neurology," pp. 22-23. Chas. C. Thomas, Springfield, Ill., 1956.

38. Penfield, W., and Jasper, H.: "Epilepsy and the Functional Anatomy of the Human Brain," pp. 61, 111. Little, Brown & Co., Boston, 1954.

39. Penfield, W., and Gage, L.: Cerebral Localization of Epileptic Manifestations. *Assn. Res. Nerv. Ment. Dis.*, 13:593-613, (Chap. 20), 1934.

40. Penfield, W., and Rasmussen, T.: "The Cerebral Cortex of Man," 248 pp., pp. 106-108, 186, 190. MacMillan & Co., New York, 1950.

41. Roberts, L.: Functional Plasticity in Cortical Speech Areas and the Integration of Speech. *Assn. Res. Nerv. Ment. Dis.*, 36:449-466 (Chap. 18), 1958.

42. Rundles, R. W., and Papez, J. W.: Fiber and Cellular Degeneration Following Temporal Lobectomy in the Monkey. *Jour. Comp. Neurol.*, 68:267-296, 1938.

43. Segre, R.: "Tratado de Foniatria." Editorial Paidos, Buenos Aires, 1955.

44. Segre, R.: Spasmodic Aphonia. *Folia Foniat.*, 3:150, 1951.

45. Smith, W. K.: The Results of Stimulation of the Uncus and Adjacent Portions of the Hippocampal Gyrus. *Fed. Proc.*, 3:43, 1944b.

46. Sugar, C.; Chusid, J. G., and French, J. D.: Second Motor Cortex in Monkey (Macaca mulatta). *Jour. Neuropath. and Exper. Neurol.*, 7:182-189, 1948.

47. Wilson, S. A. K.: Some Problems in Neurology. II. Pathological Laughing and Crying. *Jour. Neurol. and Psychopath.*, 4:299-333, 1924.

48. Wood, C. D.; Schottelius, B.; Frost, L. L., and Baldwin, M.: Localization Within the Amygdaloid Complex of Anesthetized Animals. *Neurology*, 8:477-480, 1958.

SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

The Sixth International Congress on Diseases of the Chest will be held at the University of Vienna from August 29 to September 1, 1960.

CARCINOMA OF THE TONSIL.

Surgical Treatment.*†‡

GEORGE W. ALLEN, M.D.,
and
WILLIAM GARTH HEMENWAY, M.D.,

Chicago, Ill.

INTRODUCTION.

Carcinoma of the tonsil has been given relatively little attention in the English-language literature in spite of its being one of the more common malignancies of the head and neck region. According to Martin and Sugarbaker[1] it ranks second to carcinoma of the hypopharynx in frequency among carcinomas of the pharynx. Although it has a reputation of poor prognosis, several authors[2,3,4] have recently reported impressive results of treatment, both by radiation and by surgery. The results of treatment for the largest series since 1941 are summarized in Table I. The primary type of treatment in all of these cases was some form of radiation. External radiation was mainly relied upon, but peroral and interstitial techniques were also used. Nineteen cases in the series of Scanlon, et al.,[3] had local excision of the primary in addition to radiation.

No comparable five year survival rates for surgical treatment are available. This is understandable in view of the earlier discouraging reports on the use of surgery in the treatment of this disease, but several recent reports[2,4,5] of short term results have renewed interest in this method of treatment. Klopp and Schurter[2] report nine out of 11 cases (81 per cent) free of recurrent or metastatic disease one year or more after surgical treatment. They employ an incision

---

*Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Inc., Chicago, Ill., Jan. 18, 1960.

†From the Section of Otolaryngology of the University of Chicago.

‡This investigation was supported by a special traineeship (BT-356) from the National Institute for Neurological Diseases and Blindness, Public Health Service.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Dec. 16, 1959.

which splits the lower lip in the midline. Mandibulotomy is performed, also in the midline, if necessary for exposure. For cases without palpable nodes, an upper neck dissection with excision of the primary is performed *en bloc*. For cases with palpable nodes a complete neck dissection is performed. The reconstruction employs a pedicle flap from the tongue to reconstruct the palate and pharynx. Only one of these cases had palpable cervical metastases originally. They also reported ten cases followed for less than one year with recurrences in only one. Five of these cases had cervical metastases before treatment.

Barbosa[4] reported ten cases treated by similar *en bloc* excision including partial mandibulectomy, but only four had

TABLE I.

Results from Other Centers.

Author	No. Cases	Overall Results per cent 5 yr. Survivals	No Cervical Nodes per cent 5 yr. Survivals	Cervical Nodes per cent 5 yr. Survivals
Martin and Sugarbaker[1]	148	18%		
Walker and Schulz[5]	90	15	25%	9%
Teloh[6]	162	7.2	20	4
Sheline, et al.[7]	82	20	33	9
Scanlon, et al.[3]	46	43	57	32

been followed longer than one year. Of these four cases, two were free of recurrent carcinoma.

It has been our impression that carcinoma of the tonsil almost invariably recurs within one year if it does so at all. One of our main purposes is to examine this hypothesis. Our case material consists of 64 cases of carcinoma originating in the tonsil. All were treated at least one year ago. Almost half, 27 cases, have been treated within the past five years, and follow up for the early years was not good enough for meaningful five year survival statistics.

CLINICAL DATA.

Of the 64 cases, 56 were male and eight female. Ninety

per cent of those for whom habits are recorded smoked, and 78 per cent drank alcoholic beverages.

Sore throat was the earliest symptom in 34 cases. The average duration was 4.2 months with a range of one-half to 12 months. Presence of a visible or palpable lump in the neck was the earliest symptom in 15 cases. Average duration of this symptom was 8.5 months with a range of one to 36 months. Three cases were discovered on examination for unrelated problems. Four cases could not be distinguished grossly in spite of strong suspicion based on the presence of cervical metastasis. In these the primary was demonstrated by multiple small biopsies in one case and by excision of the suspected tonsil in three cases.

TABLE II.

Age Distribution.

Age in Years	Cases
40 - 50	6
50 - 60	22
60 - 70	26
70 - 80	9
80 - 90	1
Total	64

The distribution according to age is shown in Table II.

The histological type was judged to be squamous cell carcinoma in 60 cases and transitional cell carcinoma in four cases. There were no adenocarcinomas. Sarcomas and lyphomas were excluded from this series.

CLASSIFICATION.

A system of stage grouping of these lesions is highly desirable in comparing results of treatment. The staging system proposed here is patterned after that described for the larynx by the International Committee for Stage-grouping in Cancer and for the Presentation of the Results of Treatment of Cancer[9] in 1953.

Stage I.—Carcinoma localized to the tonsil.

Stage II.—Carcinoma extending into the immediately adjacent tissues—soft palate, tonsil pillars, and tongue. No nodes palpable.

Stage III.—Carcinoma with local extension beyond Stage II or nodes palpable but not fixed.

Stage IV.—Involvement of skin, fixed nodes, or distant metastases.

Unfortunately, many cases were not described adequately enough to distinguish between stages III and IV, so an "unclassifiable" category has been added to the Tables. Our classification corresponds to that of the Mayo Clinic group[3]

TABLE III.

Classification of Cases According to Stage.

	No.
Stage I	1
Stage II	16
Stage III	25
Stage IV	11
Unclassifiable	11
Total	64

73% with Cervical Metastasis.

for stage I and II. Their stage III includes both of our stages III and IV. Their 46 cases were broken down as follows: stage I, 11 cases; stage II, 10 cases; stage III, 25 cases. Our stage distribution is given in Table III.

METHODS OF TREATMENT.

Radiation. A variety of treatment techniques were employed, but external irradiation has been the primary technique. Most patients before 1953 were treated at 250 KV. Since 1953 most cases have been treated with supervoltage radiation. The time-dose relationship was approximately 5500-6500 *r* in about six weeks. In ten cases some form of interstitial irradiation was used as an adjunct to other therapy. Eleven cases had some previous treatment elsewhere.

Surgery. Eight patients had a radical neck dissection to

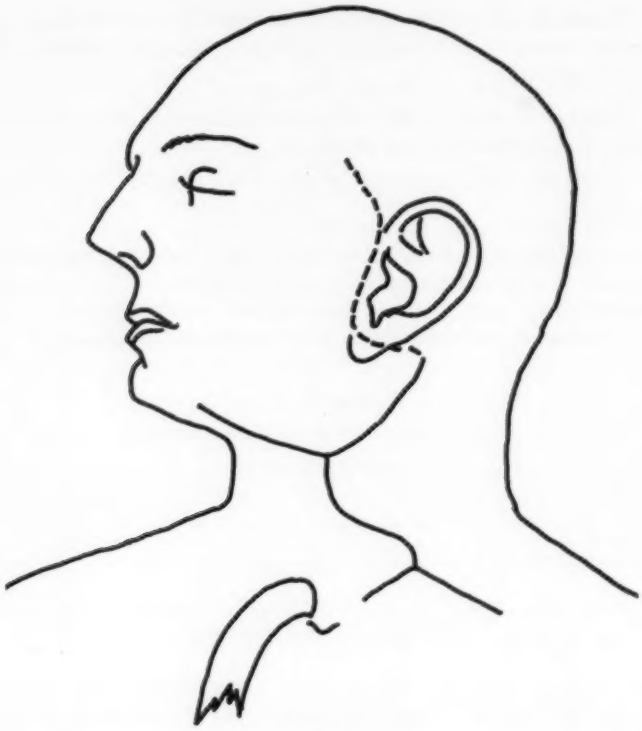


Fig. 1.

supplement external irradiation. Five patients had local excision of the primary lesion. In four cases this was supplemented by external irradiation.

Twelve cases were treated by *en bloc* excision of the primary, plus neck dissection. A basic technique has been evolved which can be varied to meet the requirements of the situation. This is outlined in the following paragraphs:

A preliminary tracheotomy is usually performed. A modified double-Y incision is then made. This features a relatively large upper flap which extends below the hyoid bone and will completely cover the pharyngeal suture line. The middle

branch is undulated, so that the incisions all meet at equal angles and the carotid vessels are crossed at right angles (see Fig. 1). We believe this incision to have several important advantages: *1.* Sharp corners are avoided; *2.* the size of the flaps is balanced; *3.* healing does not produce "checkrein" scars, and *4.* it may be continued upward in front of the ear in order to provide maximum exposure of the infratemporal fossa. The platysma is usually included in the skin flaps. The neck dissection is performed from below and behind in standard fashion, to remove the sternocleidomastoid muscle, the internal jugular vein and all of the contents of the posterior cervical triangle and the infrahyoid portion of the anterior triangle. The anterior belly of the digastric is then divided at its insertion, Wharton's duct is divided, the external maxillary vessels are ligated and divided, and the fascia colli is incised along the lower border of the mandible. The trunk of the facial nerve is dissected out. The mastoid insertion of the sternomastoid muscle, upper end of the internal jugular vein, posterior belly of the digastric and stylohyoid muscles, and the tail of the parotid gland are all dissected out and divided. The hypoglossal nerve is then dissected out well forward into the tongue and retracted downward with tape. The external carotid artery is ligated and divided and the internal carotid and vagus nerve retracted posteriorly with tape. The dissection is now varied in one of three ways depending on the location of the primary and its size.

First, if the primary is small and does not invade the soft palate, the pharynx is entered at the base of the tongue and the incision continued upward behind the posterior pillar of the tonsil. If the growth is invading the base of the tongue, the tongue is incised at a distance of 2.5 cm. around the mass. In any case the incision is made in front of the anterior pillar, and in joining the posterior incision some tongue is removed. The internal pterygoid muscle is stripped from the mandible. This and all the previous dissection is done from the outside. The final division of the soft palate and internal pterygoid muscle is then performed through the open mouth.

Second, for primary lesions invading the soft palate but not fixed and well removed from the mandible, the mandible is divided in a fish-tail cut with the oscillating saw just in front of the masseter muscle. The ramus of the mandible is then retracted laterally by stripping away the insertion of the internal pterygoid muscle. Excellent exposure can be obtained in this way with several advantages over the lip splitting incisions: *1.* Less mandible is exposed to possible infection; *2.* collateral circulation from the other side to the

TABLE IV.

Overall Results of Radiation Therapy.

	Total No.	Failures No.	Failures Per cent
All cases treated by radiation	54	48	89
Radiation 5000 *r* or more	35	29	83

TABLE V.

Results of Treatment with Radiation Alone.

Stage	Total No.	Failures Within 0-1 yr.	Within 1-2 yrs.	Lost to Follow-up	Living Without Recurrence 2-3 yr.	3-4 yr.	4-5 yrs.	5 yrs.
I	1	0	0		1			
II	11	3	1	3	1			3
III	9	6	0	2		1		
IV	8	6	0	2				
Unclassified	8	8	0					
Total	37	23	1	7	2	1		3

84% Failures.

upper skin flap is not disturbed; *3.* if removal of the ramus of the mandible appears necessary as the dissection proceeds, then as much of the body of the mandible as possible can be conserved with good blood supply, much more than if the mandible had already been divided near the midline.

Third, for very large lesions the incision can be extended in front of the ear, the parotid gland mobilized, and working first above and then below the gland, the ramus of the mandible, the zygomatic arch and the entire contents of the infratemporal fossa can be removed in the manner described by Conley.[10] With either of the two more extensive operations

the pterygoid plates are usually removed, and if necessary the maxillary tuberosity and part of the hard palate may be chiseled off. In the first and third types of operation a linear closure of the mucosa is possible. In the second type a pedicle flap of tongue is cut and rotated, to be sutured into the remaining soft palate. This is made large enough to provide good coverage for the mandible, which is then reunited

TABLE VI.

Results of Irradiation to Primary Plus Neck Dissection.

Stage	Total No.	Failures Within 1 yr.	Lost to Follow-up
II	1	1	
III	3	3	
IV	2	2	
Unclassified	2	1	1
Total	8	7	1

100% Failures.

TABLE VII.

Results of Irradiation Plus Local Excision.

Stage	Total No.	Failure	
		0 - 1 yr.	1 - 2 yr.
II	1		1
III	1	1	
Unclassified	2	2	
Total	4	3	1

100% Failures.

by criss-cross wiring. The mucosa is closed with a continuous inverting suture of 000 chromic catgut reinforced by a layer of interrupted sutures of 000 chromic catgut in the muscle and fascial layers. The skin is closed after insertion of drains. A tracheotomy tube is inserted and a pressure dressing applied.

RESULTS.

The results of treatment for all cases in which radiation was the primary method of therapy are given in Tables IV,

V, VI and VII. This includes all cases treated at the University of Chicago Clinics, regardless of whether or not therapy was initiated elsewhere. Failures are defined as: *1.* Cases showing no response to treatment, or *2.* cases showing resumption of growth after an initial response, or *3.* cases dying with carcinoma present, or *4.* cases lost to follow up. Most cases showed at least some favorable response to X-ray, but the exact number is impossible to determine. No cases lost to follow up were free of carcinoma when last seen.

TABLE VIII.

Results of En Bloc Resection.

Stage	Total No.	Failure Within 1 yr.	Living Without Recurrence	
			1 - 2 yr.	2 - 3 yr.
II	1	1		
III	10	5	4*	1
Total	11	6	4	1

55% Failures.
Five Cases were Recurrences Following Radiation,
Two Living Without Recurrence.

*One case had prophylactic radiation following surgery.

It is obvious from Table IV that even for the group treated with more than 5000 *r*, the most that the five year survival rate could be is 17 per cent.

It can be seen from Tables V, VI and VII that all recurrences, except two, that have appeared to date, have been within the first year. All cases of recurrence following surgery have been within one year. This suggests that the number of cases surviving for one year without recurrence may furnish a good standard for comparison of results of treatment by radiation and by surgery. It should be noted that only one case in which there were abnormal cervical lymph nodes responded to radiation. Radical neck dissection and local excision used as adjuncts to radiation were completely ineffective. Interstitial radiation controlled a recurrence following external radiation in one case, and this case is listed on Table VI only as a survival without recurrence.

In the nine other cases interstitial radiation was not successful in controlling recurrences.

Surgery. We have not yet treated any early lesions primarily by surgery. The one stage II case had very extensive local spread. Table VIII shows the results of surgery. Two patients died during the early postoperative period; one of hepatic failure at three months and the other of an acute, diffuse hemorrhagic gastritis at one week.

One case had local excision only and died within one year. Three cases were not treated, being seen for opinion only.

Complications of Surgery. Two patients developed large fistulae. One occurred in a patient who had surgery for a recurrence following radiation therapy. This fistula was closed successfully in two stages by a lined pedicle flap. Swallowing and speech remain unsatisfactory in this case. Swallowing and speech are practically normal in the other cases.

DISCUSSION OF RESULTS.

The tendency for early recurrence following either radiation or surgery seems clear. Of all cases seen to recur, only two have done so one year or more after treatment. This makes the early results reported by Klopp and Schurter[2] look promising. Although we have not had experience with these early lesions, our experience in cases with cervical node involvement leads us to advise surgery as the primary method of treatment in these cases. When cervical metastasis is present initially, the outlook with treatment by radiation is poor. Out of 40 of our cases with cervical metastasis treated by radiation, only one remains free of carcinoma. This was not a proved metastasis, but was described as a hard node one centimeter in diameter by a reliable observer.

Since the primary lesion often responds well to radiation, our original approach was to radiate the primary and nodes and to follow this by neck dissection when necessary. All eight of our cases treated in this way were failures. It is now our policy, when the patient has already received radia-

tion, to remove the region of the original primary together with the neck dissection *en bloc*, although there may be no gross tumor in some parts of the primary region.

Our early success and that of others[2,4] with this more radical surgery has been encouraging. Five cases out of 11, 45 per cent, have survived longer than one year without recurrence. All were advanced cases and required extensive resections, but there is little residual disability except in one case. The outlook for functional rehabilitation is better than that of cases treated by surgery for carcinoma of the larynx or tongue.

In spite of the accessibility of the tonsils to examination, there seems to be little hope for diagnosing more of these lesions before metastasis has occurred, because of the tendency to early metastasis and the relatively prolonged asymptomatic period. Many of these lesions are of small size and are very inconspicuous at a time when the cervical lymph nodes are quite obviously the site of metastatic disease. As in the nasopharynx early metastasis is probably a result of the intimate contact between the epithelium of origin and the lymphoid tissue. Careful examination will reveal the primary in many of these cases and should always include retraction of the pillars and palpation. In the presence of nodes without an obvious primary the tonsil should be removed and serially sectioned for microscopic examination.

SUMMARY.

Techniques are described for the surgical treatment of carcinoma of the tonsil. Our case material shows these neoplasms to have a very strong tendency to recur within one year, if they do so at all. For this reason we feel justified in comparing the results of radiation and surgery for cases followed for one year or longer. For 40 cases with enlarged cervical nodes treated by radiation there was only a 2.5 per cent rate for survival without recurrence. For 11 similar cases treated by definitive surgery, 45 per cent are alive and free of carcinoma. Because of this experience, we now advise surgery

as the primary method of treatment in all cases of carcinoma of the tonsil with involvement of the cervical nodes.

REFERENCES.

1. Martin, H., and Sugarbaker, E. L.: Cancer of the Tonsil. *Amer. Jour. Surg.*, 52:155-196, 1941.

2. Klopp, C. T., and Schurter, M.: The Surgical Treatment of Cancer of the Soft Palate and Tonsil. *Cancer*, 9:1239-1243, Nov.-Dec., 1956.

3. Scanlon, P. W.; Gee, V. R.; Erich, J. B.; Williams, H. L., and Woolner, L. B.: Carcinoma of the Palatine Tonsil. *Amer. Jour. Roentgenol.*, 80:781-786, Nov., 1958.

4. Barbosa, J. F.: Cancer of the Retromolar Area. *Arch. Otolaryngol.*, 69:19-30, Jan., 1959.

5. Walker, J. H., and Schulz, M. D.: Carcinoma of the Tonsil. *Radiology*, 49:162-167, Aug., 1947.

6. Teloh, H. A.: Cancer of the Tonsil. *Arch. Surg.*, 65:693-701, Nov., 1952.

7. Sheline, G. E.; Jones, M. D., and Morrison, L. F.: Radiation Therapy for Cancer of the Tonsil. *Amer. Jour. Roentgenol.*, 80:775-780, Nov., 1958.

8. Raven, R. W.: Combined Cervico-buccal Partial Pharyngectomy for Cancer of the Oro-pharynx. *Brit. Jour. Surg.*, 39:503-505, May, 1952.

9. Seventh International Congress of Radiology, Copenhagen, 1953. *Acta Radiol.*, Sup., 116, 1954.

10. Conley, J. J.: Surgical Approach to the Pterygoid Area. *Ann. Surg.*, 144:39-43, July, 1956.

SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY OF SOUTHERN MEDICAL ASSOCIATION.

The Section of Ophthalmology and Otolaryngology of the Southern Medical Association announces that they are now accepting papers by physicians of either specialty living in the area of the Southern Medical Association for consideration for presentation at the next annual meeting to be held in St. Louis, Mo., from October 31 to November 3, 1960.

The paper or an abstract of the paper may be sent directly to the Secretary, Dr. Albert C. Esposito, Suite 1212, First Huntington National Bank Building, Huntington, W. Va., as soon as possible.

THE USE OF THE RHINOTOMY OPERATION IN NASAL SURGERY.*†

JOHN E. BORDLEY, M.D.,
and
JERRY CHERRY, M.D.,

Baltimore, Md.

Since September, 1948, some 40 rhinotomy procedures have been carried out at Johns Hopkins Hospital on patients of the Otolaryngological Service. This particular technique, which was first described in 1949,[1,2] is somewhat similar to that used by Bruns in the latter part of the last Century.[3,4] It was designed to afford better access for the surgical removal of extensive lesions occurring within the nasal and paranasal spaces, while preserving to a great extent normal nasal physiology.

Inadequate exposure has long been recognized as a limiting factor in nasal surgery where extensive lesions were encountered. Each year in every large clinic three or four cases with extensive intranasal and paranasal lesions are seen, which are difficult to treat adequately by the usual intranasal techniques.

In the procedure, adopted by our clinic, the nasal pyramid is swung to one side using the soft tissues of the naso-facial angle as a hinge (see Fig. 1). This is accomplished by means of a bilateral osteotomy and an incision crossing the bridge of the nose in the region of the naso-frontal suture extending down one naso-facial angle and around the ala nasi. A second incision is made across the base of the columella. The septum is then divided with scissors on a same plane as the lateral incision of the nose (see Fig. 2). After the nose is swung aside, a submucous resection is carried out on the intranasal septal

*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., New Orleans, La., Jan. 15, 1960.

†From the Department of Otolaryngology, Johns Hopkins University School of Medicine, Baltimore 5, Md.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 22, 1960.

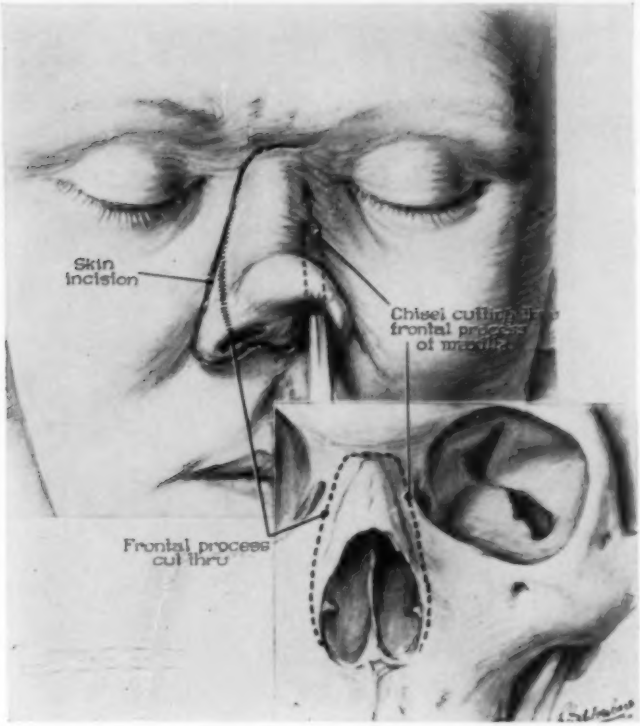


Fig. 1.

cartilage and perpendicular plate of the ethmoid bone, care being taken to leave a small anterior column of septal cartilage for later anterior support of the nose. With the nasal pyramid removed, one has a much more direct approach to the nasal sinuses. By retracting the soft tissues at the nasal-facial angle, laterally, the anterior wall of the maxillary sinus can be exposed. By freeing up the periosteum of the orbit the lamina papyracea can be visualized as well as the more medial ethmoid cells in the nose itself (see Fig. 3). By extending the transverse portion of the incision across the bridge of the nose up over the brows, both frontal sinuses can be easily ap-

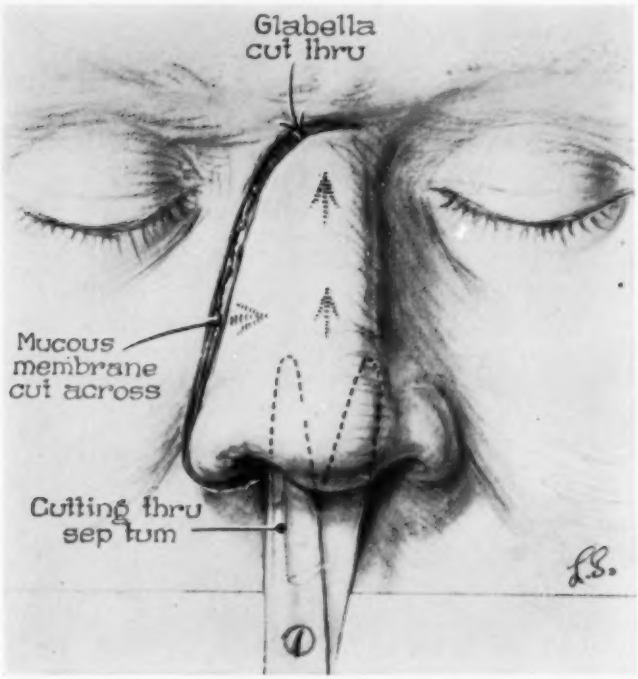


Fig. 2.

proached. The distance is shortened to the anterior wall of the sphenoid sinus and with an easily movable septum an excellent exposure of the sphenoid is obtained.

The closure after this approach is quite simple. The most important step is to resuture and realign the intranasal and pyramidal portion of the septum. This is done by placing interrupted sutures in both portions of the septum before the pyramid is swung back in place. The nasal incisions can be closed in two layers, as can the secondary incision over the frontal sinuses. In all of our cases, where removal of the septum or a portion of the septum was not required, we have had complete restoration of the septum with no postoperative perforations. This is very important in those cases where a

large portion of the lateral wall of the nose has had to be removed, as it considerably cuts down on subsequent crusting and discharge.

Care should be taken to keep any pressure of instruments or drapes away from the hinged pyramid during the operative procedure, and the pyramid should be kept wrapped in gauze

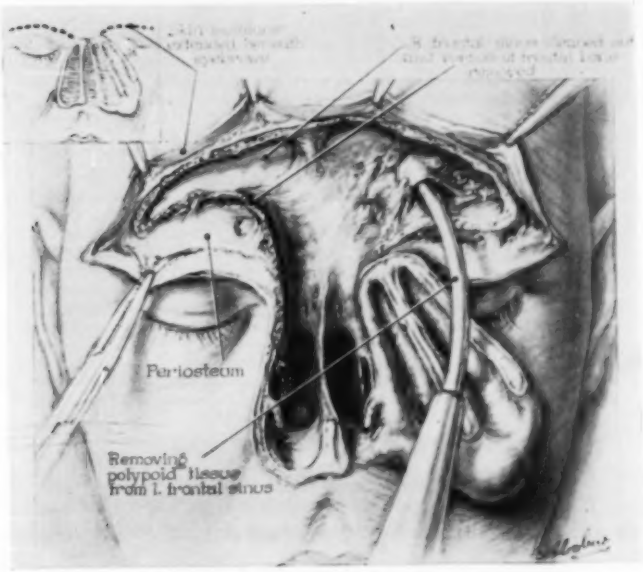


Fig. 3.

soaked in warm Ringer's solution or normal saline until it is replaced in its normal position.

Forty patients have undergone this procedure since 1947. For brevity of presentation they have been grouped into three classifications: *1.* those patients suffering from long standing chronic sinusitis (see Fig. 4) who have undergone numerous intranasal surgical procedures with resultant loss of intranasal land marks, severe scarring and degenerative

changes in the membranes of the nose. Some of these patients have developed local osteomyelitis as a result of long standing infection; *2.* the second classification covers those patients suffering from benign cysts or benign growths (see Fig. 5) within the nasal spaces or paranasal spaces; *3.* the third classification consists of those individuals suffering from malignant disease of the nose and paranasal sinuses (see Fig. 6).

INFECTION.

	Number
A. Chronic sinusitis with obstructive polyps and scarring following repeated intranasal surgery	7
B. Osteomylitis associated with chronic sinusitis	4
Total	11

Fig. 4.

BENIGN CYSTS AND TUMORS

	Number
A. Chromophobe Adenoma of pituitary gland	3
B. Retention Cysts	2
C. Encephalocele	3
D. Osteofibroma	1
E. Angiofibroma	1
F. Hemangioma	1
G. Embryonal Angioma	1
Total	12

Fig. 5.

It would be well to point out several particularly interesting cases in which we have found this type of surgery to be extremely helpful.

In two patients suffering from chromophobe adenomas of the pituitary gland, this approach was used to reach the tumor from below. One of these patients had previously had two intracranial operations and had lost the sight of one eye. This patient subsequently had to have a second operation on the adenoma and this was done through a secondary rhinotomy approach. He has remained well for the past six years. After the two operations his septum is still intact.

Recently a chrondrosarcoma involving the ethmoids, the

sphenoid and the lateral nasal wall was operated upon. It was possible to do a block resection which included the orbital floor from just lateral to the infra-orbital foramen, the lateral nasal wall, the ethmoid complex including the lamina papyracea, a portion of the cribriform plate and the anterior wall of the sphenoid. The tumor was so extensive that one would hardly believe that a cure was accomplished; however, the patient has been symptom free for the past six months.

Another case was that of a hemangioma in a boy 14 years of age involving the ethmoid complex, the middle turbinate and the medial and superior wall of the orbit. This tumor was so extensive and so close to the eye that irradiation was

MALIGNANT TUMORS OF THE NOSE AND PARANASAL SINUSES.

	Number
A. Adeno Carcinoma	4
B. Squamous Cell Carcinoma	2
C. Basal Cell Carcinoma	1
D. Papilloma (Low grade malignancies?)	4
E. Plasmocytoma	1
F. Lethal Midline Granuloma	4
G. Chondrosarcoma	1
Total	17

Fig. 6.

contraindicated. It was removed through a rhinotomy some five years ago, and to date there has been no recurrence. The patient's visual apparatus is normal and there is very little external deformity.

Of particular interest were three cases requiring rhinotomy suffering from encephaloceles. An encephalocele consists of brain tissue which has grown through some defect in the calvarium and extends into the neighboring structures. As the brain grows, the brain tissue in the encephalocele grows, displacing and destroying adjacent tissues just as a benign tumor would do elsewhere.[5,6,7,8,9,10,11,12,13,14,15]

There are some 50 nasal encephaloceles reported in the literature since 1900. Statistics show that the instance of encephaloceles are about one in every 4,000 births. They seem

to be non-hereditary, non-familial and not associated with other congenital defects. About 15 per cent of encephaloceles described are anterior and 85 per cent are occipital in presentation. The surgical approaches that have been listed are: *a.* intranasal; *b.* external ethmoid; and *c.* craniotomy. Prior to the advent of the antibiotics, the death rate following attempts to remove these tumors was extremely high.

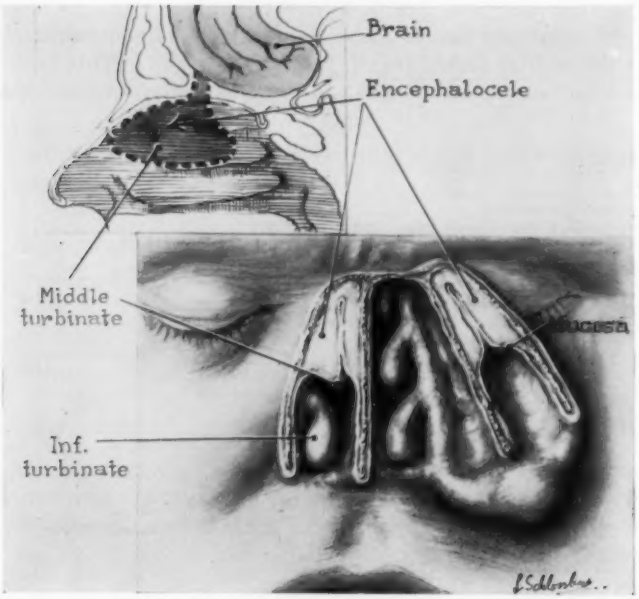


Fig. 7.

To cure such growths, the classic procedure has been to sever the stalk of the tumor at the surface of the brain by an intracranial approach, then to close the defect in the skull. Following such intracranial surgery, the mass will shrink down and in some instances apparently disappear. Under certain circustances, however, the extra cranial brain tissue will parasite a blood supply and begin to grow again. Such

growths can become locally quite destructive. This is particularly true in individuals who have not reached maturation, and where growth is still taking place in the brain itself. The excellent blood supply to the paranasal area offers an optimum opportunity for such growth. In cases where continued growth is probable or in situations where the mass of the encephalocele intereferes with normal physiological function, removal of the extra cranial growth is indicated.

Fig. 8.

In the face, the most common pathways of extension of encephaloceles are: *1.* the naso-frontal, in which the growth extends between the nasal and frontal bones destroying a portion of the nasal bones and producing a swelling at the nasal root; *2.* the naso-orbital, where the brain tissue pushes through the lacrimal-frontal suture line and produces a swelling near the inner canthus of the eye; *3.* the naso-ethmoidal, where the defect lies between the nasal, frontal and ethmoidal bones. This permits growth downward into the nose, which involves the turbinates and the septum, as well as the nasal vault; *4.* extension downward through a defect in the cribiform plate (see Fig. 7). As all of these growths enter the

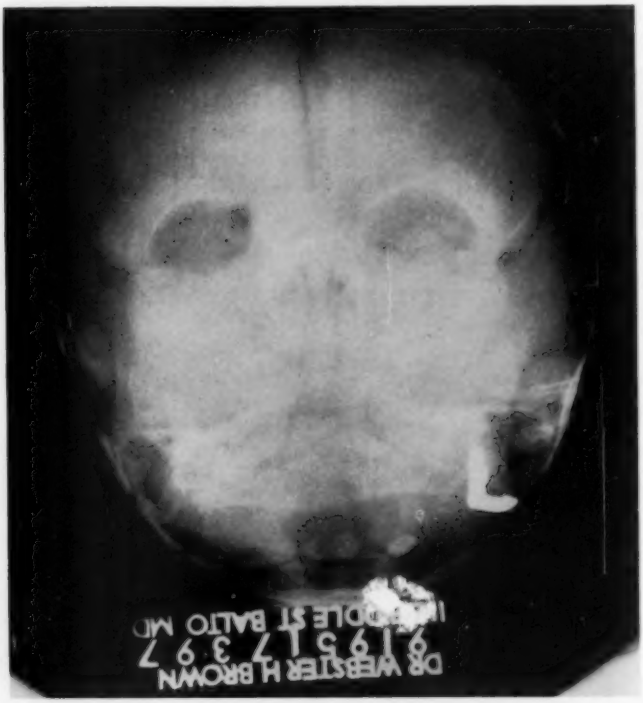


Fig. 9.

sinuses and nose from outside of the nasal spaces, they present in the nasal cavity as submucosal swelling.

In order to remove those encephaloceles that interfere with normal nasal function, it is necessary to get a good exposure of the growth. Such exposure may be obtained by using the rhinotomy approach. The three cases presented here illustrate very well some of the characteristics of these growths.

Case 1. W.G., age three months, white female (see Fig. 8—Photographs eight years postoperative).

Shortly after birth a swelling was seen about one-half a centimeter in diameter just to the right of the midline of the naso-frontal region. The mass felt slightly cystic. Examination at the age of six months showed an external mass with a diameter of about one centimeter involving the

right nasal bone, just below the level of the inner canthus of the eye. The right nostril was seen to be filled with a mass covered by normal looking mucous membranes. Breathing space was obliterated. The mass was not fluctuant. X-rays showed a defect in the right naso-frontal area (see Fig. 9). Biopsy showed brain tissue covered by respiratory mucous membrane (see Fig. 10).

At the age of six months, Dr. Earl Walker of the Neurosurgical Service, using a frontal approach, cut the stalk of the growth and closed the defect in the skull with a bone implant and a fascial graft. At nine months of

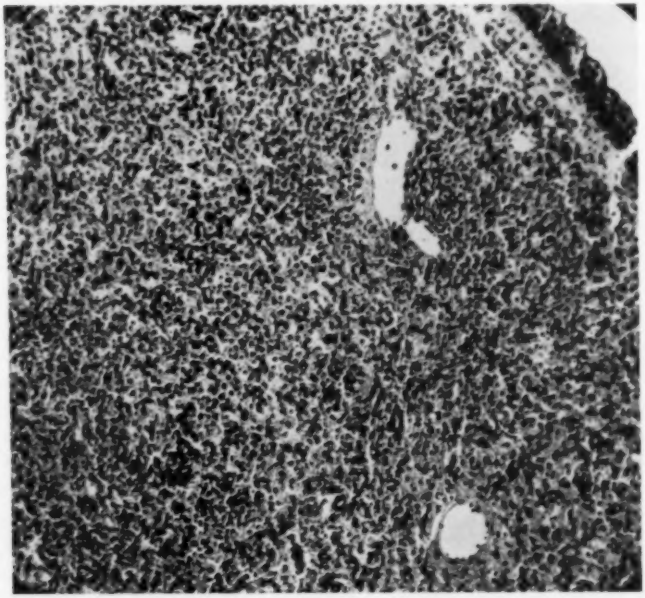


Fig. 10.

age, the nasal swelling was slightly reduced but still obstructive and a rhinotomy was performed. A relatively vascular mass was exposed filling the anterior portion of the right nostril extending down over the upper portion of the right side of the septum. It is found to have extended externally through a defect in the upper portion of the right nasal bone. This growth was entirely submucosal. Nine years later this child showed no evidence of recurrence, her breathing space is excellent, and her facial growth is normal.

Case 2. J.S., age three-and-a-half months, white male (see Fig. 11—Photographs five years postoperative).

This patient was admitted because of an obstructive mass in the right

nostril which was discovered shortly after birth. He had had two previous nasal operations before entering the Clinic. These operations were for the removal of nasal polyps. Sections made of material removed at the second operation showed brain tissue covered by respiratory epithelium. Examination disclosed an external fullness of the right side of the nose. The right nostril was filled with an edematous mucous membrane covered mass. The septum was pushed over to the left side. At four months of age a frontal lobe exploration was carried out by the Neurosurgical Service. The stalk of the tumor was found to extend through a defect about 4 mm. in diameter in the right cribiform plate. This was severed, the defect was closed with a bone implant and covered with dura from the crista galli. The mass decreased rapidly in size, the breathing space improved, and the nose became symmetrical; how-

Fig. 11.

ever at the age of four years the mass began to increase again and once more filled the right nostril. Through a rhinotomy approach the mass was found to extend over the vault of the nose and to involve the upper half of the nasal septum. The mass extended inferiorly on the lateral wall to the level below the middle turbinate. Superiorly, it extended to the cribriform plate. The right leaf of the septum was removed with the specimen. Postoperatively the patient has continued to do well. Three years after operation the breathing space is good. There is no nasal crusting, and the septum is intact.

Case 3. E.S., age 12 years, colored female.

This patient was admitted complaining of bleeding from the left nostril. Examination showed a mass involving the vault of the left nostril, the left upper lateral cartilage and the septum. The biopsy report was "necrotic tissue and inflammation, possible lethal midline granuloma." A rhinotomy revealed a growth involving the whole upper left nostril with destruction of the upper septal cartilage and a portion of the ethmoid plate. Frozen sections showed brain tissue covered with respiratory

epithelium. A stalk was exposed which extended through a defect in the left cribriform plate. This was removed from below, and the defect was closed from below with a flap of mucous membrane and a gelfoam pack. The neurosurgeons decided not to do an intracranial exploration as the brain had reached its full growth. The patient has been symptom free for the past three years. Breathing space is satisfactory and the nasal septum is intact.

As to cosmetic results, they are acceptable. Of course, those individuals losing the anterior wall of their frontal sinuses will suffer at least temporary deformity of their brows. It is possible, if the patient desires it, to later rebuild the brow with bone chips. If care is taken to follow the facial lines carefully with the original incision, satisfactory cosmetic results should be achieved.

CONCLUSIONS.

Some 40 rhinotomies have been performed at Johns Hopkins since 1947. Each patient has been carefully selected for this procedure. Lesions that do not involve the lateral portions of the maxilla must be chosen, as this surgical approach is not satisfactory in such instances. All the lesions presently reported would have been extremely difficult to expose and remove by other less deforming approaches. The operation has the advantage of maintaining the septum which helps in restoring a relatively normal nasal physiology, postoperatively. The procedure is time consuming, but has shown little tendency toward shocking the patient. The rhinotomy has a place in modern nasal surgery but should be limited to specially selected cases.

BIBLIOGRAPHY.

1. Bordley, J. E., and Longmire, W. P.: Rhinotomy for Exploration of the Nasal Passages and the Accessory Nasal Sinuses. *Ann. Otol., Rhinol. and Laryngol.*, 58:1011-1023, Dec., 1949.*

2. Bordley, J. E., and Linthicum, F. H., Jr.: Clinical Evaluation of the Rhinotomy Operation. *Jour. Kentucky State Med. Assoc.*, Dec., 1957.

3. Bruns, P.: Eine neue Methode du Temporaren (Osteoplastischen) Resection du Ausseren Nase zur. Entfernung von Nasen-Rachenpolypen. *Berl. Klin. Wochenschr.*, 9:137-140; 149-153, 1872.

4. Bergmann, E.; Bruns, P., und Mikulicz, J.: "Handbuch der Praktischen Chirurgie," see Band I, Chirurgie des Kopfes, S. 983. Ferdinand Enke, Stuttgart, 1900.

*Figs. 1, 2 and 3 of the present publication are from the cited paper.

5. Anderson, F. M.: Intranasal Encephalocele; Report of Case with Intracranial Repair and a Review of Subject. *Arch. Otolaryngol.*, 46:644-654, Nov., 1957.

6. Mood, G. F.: Congenital Anterior Herniations of Brain. *Ann. Otol., Rhinol. and Laryngol.*, 47:391-401, June, 1938.

7. Gisselsson, L.: Intranasal Forms of Encephaloceles. *Acta Oto-Laryngol.*, 35:519-531, 1947.

8. Moore, P. M.: Intranasal Encephalomeningocele. Report of Case. The Laryngoscope, 62:659-677, July, 1952.

9. McGillicuddy: Encephalomeningoceles in Nasal Cavities. *Ann. Otol., Rhinol. and Laryngol.*, 51:516-526, June, 1942.

10. Cassidy, W. A., and Wahl, J. W.: Congenital Displacement of CNS Tissue in Nasal Fossa. *Arch. Otolaryngol.*, 59:93-99, 1954.

11. Fineman and Pich: Intranasal Encephalomeningocele. *Ann. Otol., Rhinol. and Laryngol.*, 62:114-120, 1953.

12. McCormick and Harris: Neurogenic Tumors of the Nasal Fossa. *Jour. A.M.A.*, 157:318-321, 1955.

13. Walker, E.; Moore, W. W., and Simpson: Intranasal Encephaloceles, Survey of Problem with Recommendation for Reducing Mortality. *Arch. Otolaryngol.*, 55:182-187, 1922.

14. Davis and Alexander: Congenital Nasofrontal Encephaloceles. *Jour. Neurosurg.*, Vol. 16, No. 4, July, 1959.

15. Dodge and Love: Intranasal Encephalomeningoceles with Cranium Bifidum. *Arch. of Surg.*, Vol. 79, No. 1, July, 1959.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES.

The 1960-1961 Home Study Courses in the basic sciences related to opthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of 10 months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 15 Second street S.W., Rochester, Minn. Registrations should be completed before August 15.

CLOSURE OF PERFORATIONS OF THE TYMPANIC MEMBRANE BY VEIN GRAFTS.

A Preliminary Report of Twenty Cases.*†

HAROLD G. TABB, M.D.,

New Orleans, La.

Early in 1959, after watching Dr. John J. Shea, Jr., of Memphis, perform his ingenious fenestra ovalis operation, I became interested in his use of vein grafts, which, he informed me, he had also used to repair lacerations of the eardrum, with good results. It later occurred to me that these grafts would be useful for the closure of large perforations of the tympanic membrane, particularly those of long standing (see Fig. 1).

By this time I, like many others, had become dissatisfied with the use of skin for this purpose, as skin grafts were hard to manage, and they were not usually an acceptable substitute for normal tympanic membrane. They often remained thick and moist, and normal processes of desquamation continued. They showed little resistance to infection, which was often followed by perforation or sloughing of the graft. There was also the constant danger of cholesteatoma formation on the under surface of the graft, from the downgrowth of squamous epithelium through one of its sectioned appendages. Dr. Harold F. Schuknecht demonstrated such a case to me in August, 1959.

With this climate for reform, I decided to test vein grafts in the repair of perforations of the tympanic membrane. The results were so gratifying that a preliminary report of our cases, which now number 20, seemed justified.

It was not until after the report was completed that I

---

*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., New Orleans, La., Jan. 16, 1960.

†From the Department of Otolaryngology, Tulane University of Louisiana School of Medicine.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Feb. 1, 1960.

learned that Dr. Shea had been working on closure of perforations of the tympanic membrane, in addition to repair of tympanic lacerations, and that he had reported a method of closure which had been successful in 13 of 14 cases.[1]

Although we had worked entirely independently, his technique is similar to one of the techniques which I have been using.

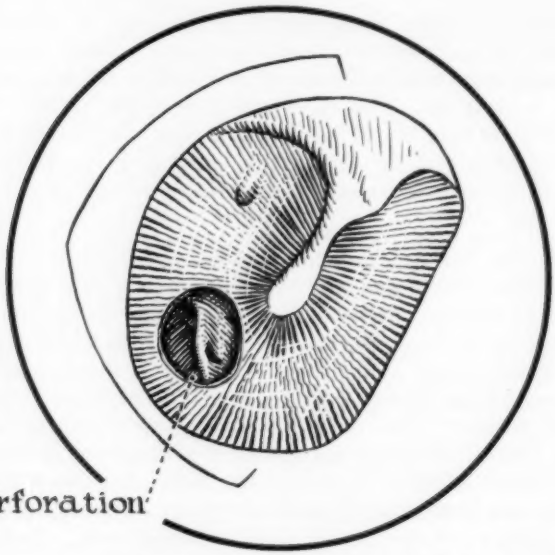


Fig. 1. Typical long-standing perforation of posteroinferior quadrant of tympanic membrane.

To date, 20 operations by this technique have been performed on our service, with a follow-up varying from two to ten months. I am presenting five of these cases in detail (see Fig. 2) because they represent the simple, persistent type of tympanic perforation, without associated middle ear or mastoid disease, which responded to this technique after they had resisted other techniques of closure. In five of the other 15 cases the perforations were closed at the time of mastoid surgery.

Preoperative examination in each of these five cases ruled out any underlying infection and demonstrated the patency of the Eustachian tube on the affected side. After an initial audiogram, a paper patch was applied to the perforation and allowed to remain *in situ* for a week or more, to ensure that the ear was dry. In each case the ear was dry, and in each case there was prompt improvement in hearing. This pre-

Patient	Age	Perforation Data: Ear	Type	Duration	Method used Previously	No. Days Healing (Inflation-Pneumomassage)	Vein Donor Site	Post-Op. Observation Period without Sequela
TH	8	AD	Central	9 mos.	Acid cautery patch	14	Foot	10 mos.
DM	7	AS	Central	7 mos.		13	Arm	9 mos.
DWM	37	AS	Central	3 yrs.	Acid cautery patch Irritant patch Myringoplasty	18	Hand	6 mos.
LL	8	AD	Marginal	7 yrs.	Acid cautery patch	12	Arm	4 mos.
CB	30	AS	Central	5 yrs.		14	Hand	2 mos.

Fig. 2. Summarized case histories of five closures of perforations of the tympanic membrane by vein grafts.

liminary use of the paper patch as a sort of therapeutic test was suggested by Dr. Robert Henner, of Chicago.

CASE REPORTS.

Case 1. T.H., an 8-year-old boy, had a 4-mm. central perforation of the right tympanic membrane which had persisted after a suppurative otitis media nine months earlier. Repeated attempts at closure by the trichloracetic acid cautery-paper patch technique had failed.

There was no underlying middle ear or mastoid disease, and the right Eustachian tube was inflatable. A 15 db hearing loss in the speech frequencies was eliminated when a paper patch was applied.

A vein graft was placed under the perforation, with the endothelial surface facing the tympanic cavity, through a stapes incision. Four days later a Staphylococcus aureus infection developed. It was controlled with specific antibiotic therapy, without evident damage to the graft. On the seventh postoperative day, numerous blood vessels could be seen crossing the tympanic membrane just under the epithelial surface and

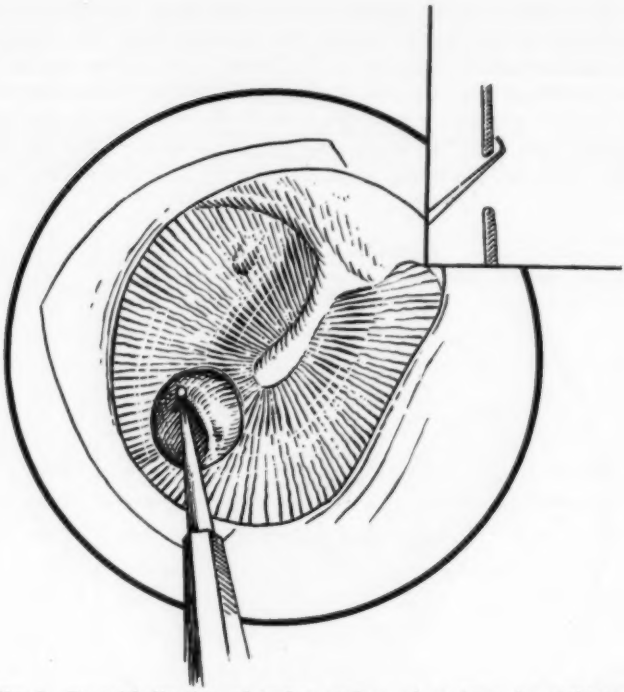


Fig. 3. De-epithelization of under surface of eardrum by right-angle knife. Insert shows detail of technique.

apparently supplying the graft. On the fourteenth postoperative day, the perforation appeared to be firmly closed. The drum was then massaged and the tympanic cavity inflated.

At the present time, 10 months after operation, the perforation has remained firmly closed, and the eardrum appears practically normal except for a whitish plaque at the site of the former opening. For three months during the Summer this ear was exposed to the hazards of swimming and suffered no ill effects although it was not protected in any way.

Case 2. D.M., a seven-year-old boy, had a central perforation, 3 - 4 mm. in diameter, of the left tympanic membrane; it had developed seven months earlier, after an upper respiratory infection and purulent otitis media. There was no underlying infection, and the ear remained dry for a month, during which time a 10 db hearing loss was eliminated.

Thirteen days after closure of the perforation by a vein graft placed through a stapes incision, healing was sufficiently firm to permit massage of the drum and inflation of the middle ear.

Swimming was allowed a month after operation. A month later, the

patient appeared with an acute external otitis, from a *Proteus* infection, which was controlled with specific antibiotic therapy. The repaired tympanic membrane was as resistant to spread of the infection as if it had been a normal structure. At the end of nine months the results are entirely satisfactory.

Case 3. D.M., a 37-year-old man, was first seen in July, 1938, with drainage from the left ear of three weeks' duration. A perforation of the tympanic membrane had been successfully repaired a year earlier by myringoplasty and the patient had had no difficulty until the present episode of otitis media. When the infection was controlled, a large

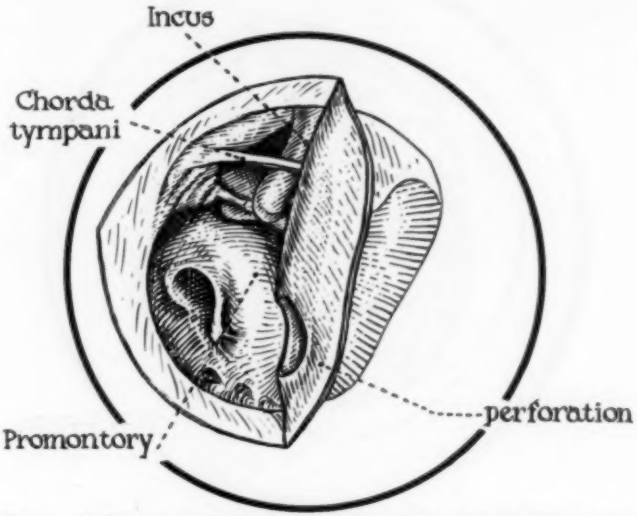


Fig. 4. Reflection of tympanic membrane, after stapes incision, to expose perforation.

kidney-shaped central perforation was left. The Eustachian tube was patent. The patient had a 10 to 15 db hearing loss.

After the ear had been dry for a month, closure was accomplished by office treatment with the acid cautery and irritant drops. At the end of five months the drum appeared normal and hearing was improved.

Three months later the patient returned with acute suppurative otitis media. After it was controlled, there was again a large central perforation, which this time did not respond to office methods of closure. In June, 1959, closure was effected by the use of a vein graft applied through a stapes incision. Healing was complete in 18 days and the patient has had no hearing or other difficulties in the six months since operation.

Case 4. L.L., an 8-year-old girl, had had a marginal perforation of the right tympanic membrane after a purulent otitis media when she was a year old. Drainage developed whenever she went in swimming. An adenotonsillectomy two years before had had no effect on the perforation,

and an attempt to close it with the acid cautery and a paper patch 18 months later also had failed. There was no underlying middle ear or mastoid disease and the Eustachian tube was patent, but there was a mild conductive hearing loss.

Under general anesthesia, a stapes incision was made, to permit examination of the middle ear structures, and a vein graft was placed under the perforation and held in place by Gelfoam.® Healing was firm 12 days after operation and at the end of four months the results, including improvement in hearing, are still good.

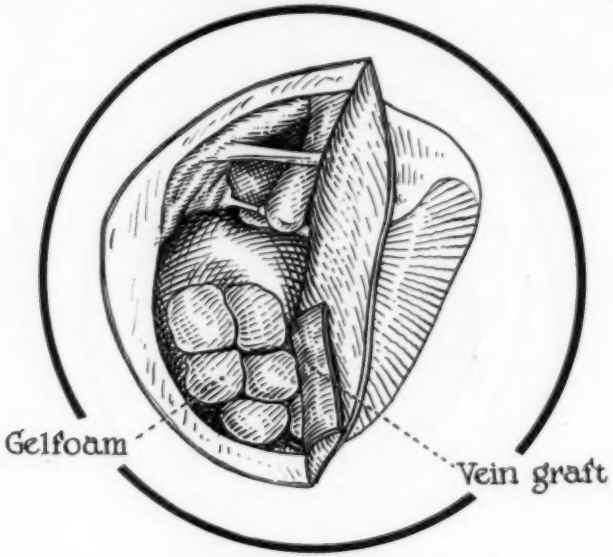


Fig. 5. Vein graft applied to under surface of tympanic membrane and held in place by Gelfoam®.

Case 5. C.B., a 30-year-old man, had had a large central perforation of the left tympanic membrane for the past five years. Suppurative otitis media developed whenever he had an upper respiratory infection. There was a conductive loss of 25 db in this ear. The Eustachian tube was patent and the ear was dry. The use of a paper patch eliminated the hearing loss.

A vein graft was placed through the central perforation and positioned satisfactorily without the use of a stapes incision. The perforation healed in 14 days, and at the end of two months the results, including the restoration of hearing to normal, are entirely satisfactory.

TECHNIQUE.

In the first cases in this series it was our practice to em-

ploy the so-called stapes incision (the incision used in mobilization of the stapes), adapted from Lempert,[2] and the vein graft was placed on the inferior surface of the drum through it. Later, it was realized that if the perforation were sufficiently large for the necessary manipulations, the incision was not necessary. Thereafter, it was omitted unless it was

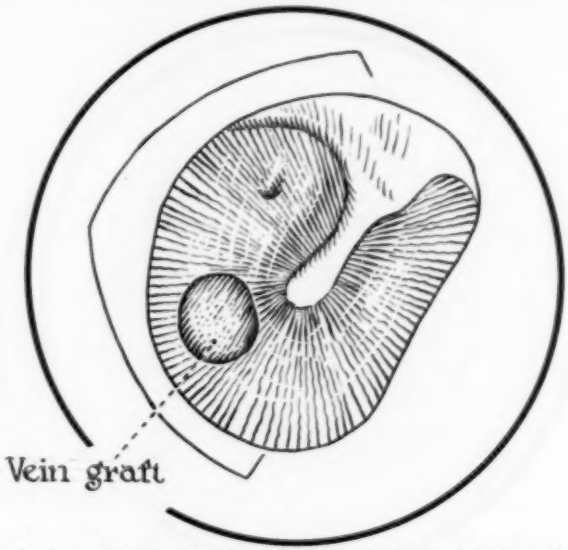


Fig. 6. Vein graft in situ after replacement of reflected tympanic membrane.

thought that exploration of the middle ear or some procedure on the middle ear would be necessary.

Whether the incision is or is not used, the initial steps of the procedure are the same:

1. The auricle and external auditory meatus are very carefully cleansed under microscopic observation.

2. Basal sedation is used before operation. The procedure is carried out under local infiltration anesthesia in adults and under general anesthesia in children.

3. The perforation of the tympanic membrane is visualized and inspected with an operative microscope with 10-power magnification.

4. A right-angle flap knife with a 2-mm. blade (see Fig. 3) is inserted through the perforation and is used to de-epithelize the under surface of the eardrum for a distance of 2 - 3 mm.

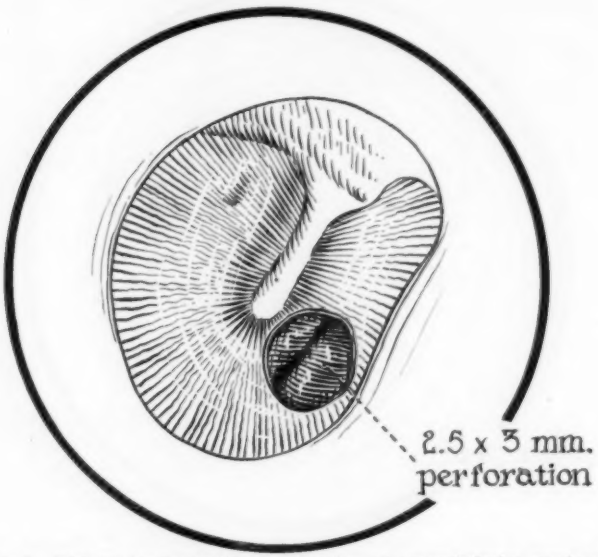


Fig. 7. Typical long-standing perforation of anteroinferior quadrant of tympanic membrane.

from the edges of the perforation, in order to create a raw vascular bed for nourishment of the vein graft.

If the technique first employed is used, a stapes incision is made parallel to the margins of the posterior half of the tympanic membrane. The tympano-meatal flap thus created (see Fig. 4) is reflected anteriorly, to expose the tympanic cavity and the under surface of the eardrum.

A tubular section of vein, usually about 1 cm. in length, is taken from the dorsum of the hand in adults, and from the

foot or arm in a child, by the Shea technique. The resected vein is sectioned vertically on a lateral aspect and is opened, thus creating a flat, rectangular graft. It is kept moist with a drop of saline solution while excess adventitia is removed under magnification.

The graft is then placed over the perforation, so that the

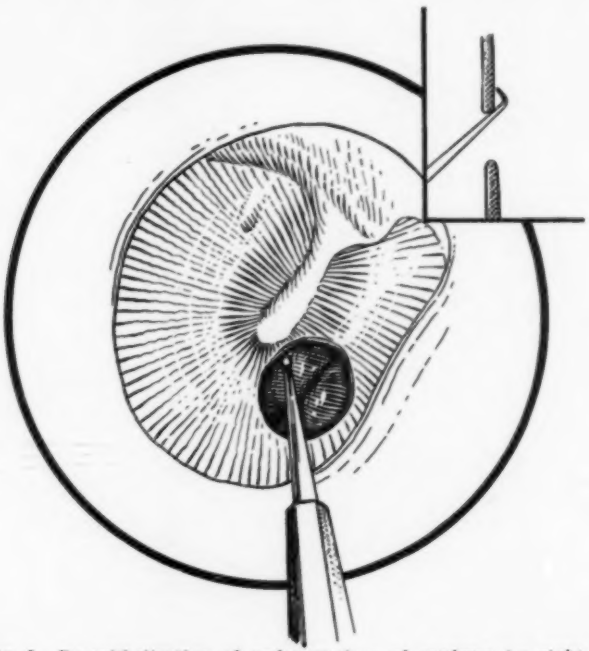


Fig. 8. De-epithelization of under surface of eardrum by right-angle knife. Insert shows detail of technique.

edges of the aperture are overlapped by it for 2 - 3 mm. The endothelial surface of the graft lies toward the tympanic cavity and its adventitial or raw side lies in apposition to the vascular bed created on the under surface of the drum. Gelfoam® is applied to hold the graft in position across the perforation (see Fig. 5). The tympanic membrane is replaced in its original position (see Fig. 6) and the external

surface of the drum is covered with Gelfoam® to a depth of about 0.5 cm.

The initial steps of the second technique, as already noted, are the same as far as the de-epithelization procedure (see Figs. 7, 8). The stapes incision is omitted, and Gelfoam® soaked in physiologic salt solution is introduced through the perforation (see Fig. 9), to ensure good contact of the graft

Fig. 9. Introduction of Gelfoam® through perforation, to ensure good contact of graft and under surface of tympanic membrane.

and the under surface of the tympanic membrane. The graft is then introduced through the perforation (see Fig. 10) and fitted to the under aspect of the drum, so that, as in the first technique, there is an overlap of 2 mm. around the edges of the perforation (see Fig. 11). It is held in place by Gelfoam,® which is also used to cover the external surface of the tympanic membrane.

In the technique devised by Dr. Shea, the vein graft is placed between the outer epidermal layer and the inner mu-

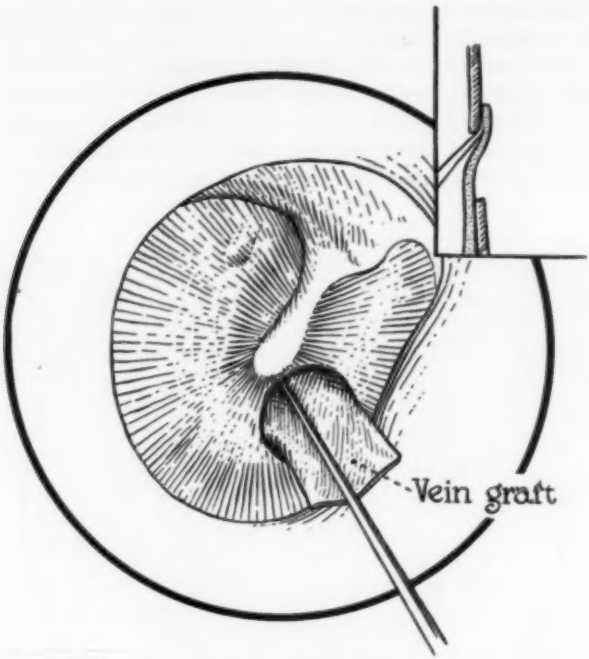


Fig. 10. Application of vein graft through perforation. Insert shows graft being tucked under edges of perforation.

cosal layer of the tympanic membrane. In all of our cases, the graft was placed either underneath the tympanic membrane or on top of it, after de-epithelization of the bed on which the graft was to rest. This technique was followed whether or not a stapes incision was used.

COMMENT.

There is no question that perforations of the tympanic membrane should be closed, to preserve aural health and function. There is also little argument that surgery is the technique of choice for all children and for most adults. It accomplishes healing in a shorter time than office methods, and the percentage of permanent good results is higher.

These facts were generally true even when skin grafts, with their undesirable features, were used to close the perforated tympanic membrane. Dr. Shea's experience, like my own, indicates that results are much better when vein grafts are used. For this there are a number of reasons. Veins (see Fig. 12-a) are thinner and more pliable than skin and, therefore, easier to work with. They can be procured in suitable

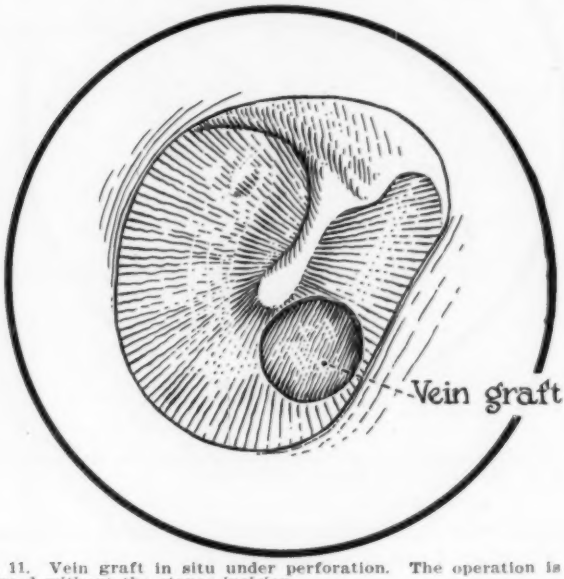


Fig. 11. Vein graft in situ under perforation. The operation is easily performed without the stapes incision.

sizes from the hand, foot, arm, or even the neck, with hairline incisions. They are easy to prepare for grafting. They contain no keratinizing epithelium. They are easily applied with an ear microscope through an incision in the canal or through the perforation itself, without an endaural or postauricular incision. They adapt well to the contours of the ear. They usually require very little contact for nourishment and will bridge areas of 7 - 8 mm. without sloughing.

De-epithelization of the under surface of the perforation

for a distance of a few millimeters away from its margin not only provides a good bed for the graft but also removes any epithelium which may have grown around the edges of the perforation onto the under surface and which would interfere with closure. The presence of epithelium in this location was advanced by Dunlap and Schuknecht[3] in 1947 as a possible cause for non-closure of these perforations.

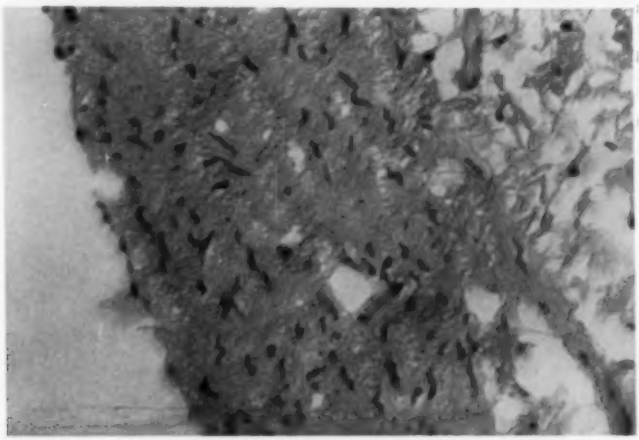


Fig. 12-a. Microscopic demonstration of vein from hand, showing endothelium, muscular coat, and adventitia.

We have demonstrated that autogenous vein grafts will grow over or under perforations of the tympanic membrane (see Fig. 12-b) and will seal perforations in old skin grafts (see Fig. 13).

Vein grafts under perforations of the tympanic membrane appear grossly to serve as tissue splints. Apparently they have the ability to extract an extra blood supply and produce an inflammatory response around the perforation, so that natural closure, by proliferation of the squamous epithelium, is stimulated.

When healing of the perforation is observed under the ear

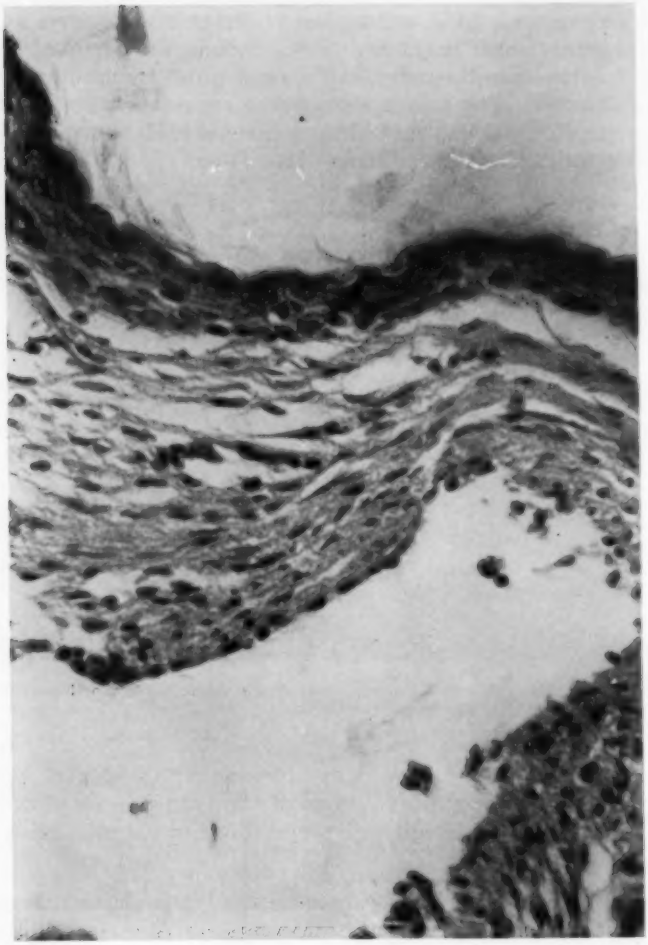


Fig. 12-b. Experimental demonstration showing healing of perforation of drum four weeks after application of vein graft. Note squamous epithelium on graft.

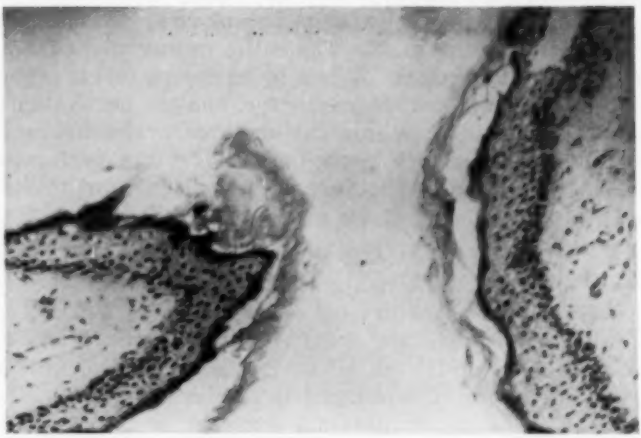


Fig. 13. Microscopic demonstration of squamous epithelium growing around edges of old perforation in full-thickness skin graft. Secondary vein graft repair was successful.

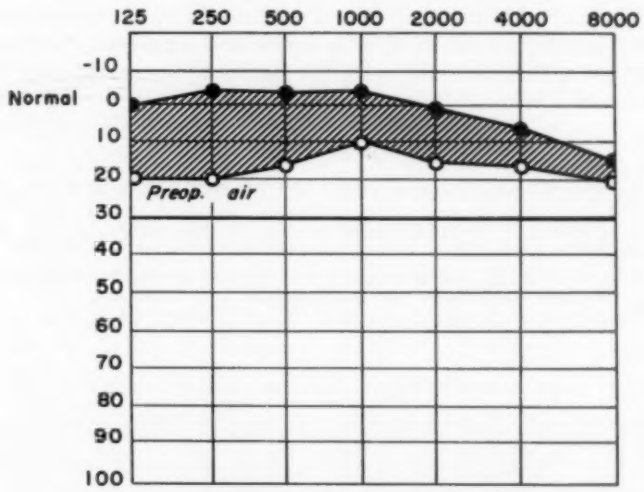


Fig. 14. Average gain in hearing in five cases of successful vein-graft closure of perforations of tympanic membrane.

microscope, within three or four days large blood vessels can be seen crossing the tympanic membrane under the epidermis and penetrating the graft. The entire membrane and graft are diffusely hyperemic. Within seven days, a seal is present between the graft and the membrane, and the perforation is perceptibly smaller. Within two to three weeks, healing is complete. The surface epithelium bridges the perforation and the vein appears as a pink, subepithelial area of thickening.

In a number of the cases in this series, as indicated in those presented in detail, the vein-grafted tympanic membrane successfully withstood the hazards of swimming as well as suppurative middle ear and external ear infections, without damage to the graft. In all of the cases treated, hearing losses were improved or completely eliminated (see Fig. 14). Dr. Shea in his report does not comment on his results in regard to hearing.

CONCLUSION.

At the present time, surgical repair of perforations of the tympanic membrane, by the Shea technique of autogenous vein grafts, seems to offer a quick and apparently certain method of closure with, at the same time, restoration of maximal hearing.

REFERENCES.

1. Shea, John J., Jr.: Vein Graft Closure of Eardrum Perforations. In press.

2. Lempert, J.: Tympanosympathectomy: A Surgical Technic for the Relief of Tinnitus Aurium. *Arch. Otolaryngol.*, 43:199-212, March, 1946.

3. Dunlap, A. M., and Schuknecht, H. F.: Closure of Perforations of the Tympanic Membrane. The Laryngoscope, 57:479-490, July, 1947.

ALLERGIC MANIFESTATIONS IN OTOLOGIC DISEASE.*†‡

BEN H. SENTURIA, M.D.,

St. Louis, Mo.

One finds many reports in the literature purporting to deal with allergic manifestations in the external and middle ear, but little basic investigative work is presented to substantiate these claims. In this presentation, some widely accepted allergic concepts dealing with the external and middle ear are considered and discussed in the light of our own experience and investigative work.[1] These problems have been reviewed recently by Hoople,[2] Koch,[3] Derlacki,[4] and Kuhn.[5]

It is necessary, first, to define my concept of the allergic state before we enter into any discussion of the role it may play in these otologic conditions. It is agreed by most workers that the original Von Pirquet definition of allergy as an "altered reactivity of the tissues" is much too broad in the light of our present knowledge; furthermore, it is difficult to accept the suggestion[6] that any phenomena which result from autonomic dysfunction without obvious antigenic stimuli or classical immunological changes should be included under the term "allergy."

I do not insist that the term "allergy" be restricted to an acquired specific alteration in the capacity to react predicated on an antigen antibody mechanism,[7] but I prefer to narrow the definition of an allergic state to those listed by Rackemann and others:[8,9]

1. A history of either past or current allergic symptoms or signs.

---

*From the Departments of Otolaryngology, Washington University School of Medicine and Jewish Hospital, St. Louis, Mo.

†This paper, in part, was presented before the section of Ophthalmology and Otolaryngology of the American College of Allergists on April 25, 1958.

‡The investigative work reported in this paper was performed with the assistance of funds from the St. Louis Otological Foundation.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Sept. 17, 1959.

2. A history of allergy in antecedents or descendants.

3. Remission of symptoms or signs on elimination and recurrence on re-exposure to specific allergen.

4. Blood or secretion eosinophilia.

5. Positive scratch, intradermal or patch tests.

6. Demonstration of circulating antibodies (passive-transfer tests).

THE EXTERNAL EAR.

Many otologists are convinced that allergy plays a major role in external otitis. With the above concept of the allergic state in mind, let us examine some of the diseases which we have listed as "allergic" in our classification of diseases of the external ear.[1,10]

A. Contact Type Dermatitis (Dermatitis Venenata).

The allergic inflammation herein produced is a result of the contact of the offending allergenic substances (animal, vegetable or mineral) with the hypersensitive epithelial cells of the epidermis. A so-called period of incubation or sensitivity varying from approximately five to 21 days is required for the production of large quantities of antibodies and the development of the hypersensitive state. The reaction consists of erythema, edema, vesiculation, bullous formation and crusting, and is accompanied by a sensation of itching, burning, stinging and pain in varying degrees. Involution occurs when the contactant is removed or neutralized. The usual substances responsible for this type of dermatitis in the ears are ear medications, cosmetics applied to the scalp and face, eyeglass temples, telephone receivers, hair lacquers, perfumes, colognes and earrings. Actually, any medication used in and around the ear is potentially capable of sensitizing the skin. Patch tests with the suspected agents are very successful in isolating the responsible allergens.

B. Atopic Dermatitis.

This is a chronic dermatitis with intense pruritis. There

are frequently associated lesions of the eyelids, face, sides of the neck, popliteal and anti-cubital fossae. The skin affected is erythematous, scaly and excoriated. Such a reaction may occur in any part of the ear, in which case the area involved often becomes thickened and inflamed with oozing patches. In about 50 per cent of the cases there is an associated history of other allergic diseases such as asthma, hay fever, migraine and urticaria. A familial history of these diseases is common. Such patients possess a high percentage of circulating antibodies in their blood serum. This disease does not respond well to the allergic approach, however, and there is thought to be some additional factor responsible for the reaction.

C. Drug Eruptions.

Morphologically the drug eruption may assume any form from simple erythema to vesiculation, papule and bullae formation. With very few exceptions, any drug given orally or parenterally is capable of producing specific sensitization. Some of the common offenders are penicillin, "sulfa" drugs, barbiturates, salicylates, bromides, iodides, arsenic, quinine, etc. As a rule, the rest of the body is also affected, but the eruption may be "fixed" to the ear alone.

D. Infectious Eczematoid Dermatitis.

In this disease there are visible on the skin of the auricle and periauricular tissues, discrete erythematous, weeping, vesiculated and crusted lesions. An adjacent focus of infection which is usually a pyoderma or a draining purulent otitis media and/or mastoiditis can be demonstrated. It is presumed that the discharge sensitizes the peripheral skin over which it passes.

E. Physical Allergy.

The ear may be affected as part of the general reaction which results from exposure to cold, heat, pressure, ultraviolet rays, or rays from the visible spectrum. The usual manifestation is urticaria, erythema or eczematization. The mechanism may be based on a true antigen-antibody reaction, the antigen being a normal occurring protein which is altered when it is exposed to the physical agent.

The largest area of difficult differential diagnosis lies in the overlapping of neurogenic and allergic dermatoses. In the neurogenic group we include simple pruritis, neurodermatitis, neurotic excoriations and artifact dermatitis. The persistent itching which is invariably present may easily be interpreted as a manifestation of an allergic reaction. If none of the postulates of an allergic state exist one should attempt to ferret out basic underlying neurogenic factors.

In simple pruritis and localized neurodermatitis a proper understanding on the part of the patient will frequently bring about rapid control of the condition. On the other hand, in many cases superficial psychotherapy by the otologist, or more profound therapy by the psychiatrist will be necessary to control properly generalized neurodermatitis, neurotic excoriations, etc. The sympathetic and conscientious allergist will provide the neurogenic patient with a much needed crutch and in many instances will give relief as a consequence of the interest and care provided; but we must not misinterpret these results as evidence for an allergic etiology when in fact we are dealing with a skin manifestation of a severe underlying psychological conflict.

In an effort to understand something of the pathogenesis of external otitis, Dr. Lawrence Sophian and the author obtained biopsies of the skin from five patients with the history and findings of acute diffuse external otitis.[11] No allergic history was elicited from any of the subjects, but four of them reported recurrent ear infections. Because of the very severe acute dermatitis, incision and drainage were required; thus it was possible to take biopsies. Serial sections were prepared.

In addition to the very acute inflammatory changes present, a differential count on the dermal infiltrate revealed an average for all specimens of 24 per cent eosinophiles (range 12 to 31 per cent). The capillary and arteriolar vessels showed dilatation, endothelial swelling, hyperplasia and sometimes thrombosis, necrosis, and vascular reactions which can be associated with allergic inflammation. These findings were at a substantial variance with what would be anticipated

in common pyogenic inflammation, and were not found in skin biopsies of patients with chronic otitis media.[12]

One might argue in retrospect that there are certain clinical and pathological aspects of external otitis which are compatible with a state of allergy. Among these are the recurrent nature of external otitis, dry skin and persistent pruritis prior to the acute exacerbation, vascular changes and a high percentage of eosinophiles. On the other hand, the mere presence of eosinophiles does not provide a definite diagnosis of allergy since these cells may be associated with many other conditions such as tissue anoxia, parasitic infections, Loeffler's disease, etc.; and it is not unusual to find an increase in eosinophiles in some inflammatory processes.

Of much significance was the recent study by Jones and Norman,[13] who reported on biopsies obtained from 20 patients with external otitis. In only two specimens was there any striking increase in tissue eosinophiles, and the authors expressed the opinion that "nothing in the histological features of the inflammatory process . . . suggest the hypersensitive mechanism."

It is interesting that 14 of their patients showed 3 per cent or more eosinophiles in the peripheral blood, and two showed a distinct increase in the number of eosinophiles present in the tissue exudate. If these patients gave a positive family history of allergy and of persistent pruritis or chronic eczema it might be desirable to consider some of these ears as allergic-suspect, and a search for further evidence of the allergic state would be indicated.

I am actually pleased that in this large series reported by Jones and Norman, only a small number of the biopsies showed a significant increase in eosinophiles. On the basis of my clinical experience with acute diffuse external otitis, I was not happy with the implications of our own histopathologic findings; however, although I do not believe that a state of hypersensitivity plays a frequent role in acute external otitis, I would urge that we keep in mind that a chronic allergic dermatosis may provide the fertile substrate in which there may be superimposed infection with Gram-negative

bacilli.[14] A picture of fulminating acute diffuse external otitis might then overshadow the basic underlying allergic state.

THE MIDDLE EAR.

Much has been written about the allergic middle ear. Unavoidably, some confusion has resulted from combining under one title of chronic otitis media, those ears with middle ear effusion and an intact drum (otitis media with effusion, secretory catarrh, catarrhal otitis) and those with a perforated tympanic membrane and chronic otorrhea.

Dohlman[15,16] and Koch[3] have written at great length regarding the frequency of allergic changes in the chronic draining middle ear. Dohlman emphasized the importance of an allergic evaluation in patients with non-responsive chronic otitis media and demonstrated eosinophiles in the secretions. Koch described in great detail the cytologic changes present in the secretion of cases with chronic otitis media with perforation. He found a significant number of eosinophiles and changes compatible with the allergic state in 19.8 per cent of his 262 cases.

Derlacki[17] described the characteristic picture of allergic chronic otitis as showing viscous or gelatinous secretion with a considerable number of eosinophiles, thickened mucoperiosteum, protracted healing occurring in allergic individuals and good response to anti-allergic treatment. He stated that in recurrent acute otitis media an infectious inflammatory process may be superimposed on an allergic tympanum and tubal mucosa. Derlacki is of the opinion that in chronic allergic suppurative otitis media with a benign central perforation, an acute infection of the tympanum either precedes or is concurrent with an allergic involvement of the tubal and middle ear mucosa.

On the other hand, Dean and Pfingsten[18] found an increase in eosinophiles in only one of 26 cases of chronic otitis media studied. Ojala and Palva[19] observed 124 cases of acute and chronic otitis media and mentioned only one instance of apparent allergic origin.

It is necessary that we clearly differentiate the chronic draining ear from that category of middle ear disease which we will refer to as otitis media with effusion. In this latter condition we are dealing with various types of middle ear effusions with an intact tympanic membrane.

Lewis[20] and Proetz[21] suggested that localized allergic edema in the tympanum could simulate acute purulent otitis media. King[22] studied 72 ears of 56 children and found that seven of the 39 ears with mucous secretion showed five to ten per cent eosinophiles, while none of the children with serous effusion had any eosinophiles. Ivstam[23] reported smears from 33 cases with secretory catarrh, and 13 of these showed eosinophiles in very small numbers.

Derlacki[4] stated that "the majority of the cases of chronic secretory otitis with either a clear serum or a much thicker mucus are due to a definite allergy to house-dust or to a food" but offers no substantiating evidence. Jordan[24] reported on 123 patients with middle ear effusion among whom 91 (74 per cent) were diagnosed as allergic on the basis of the findings of eosinophiles in nasal smears, positive skin tests and response to allergic therapy; yet Jordan found that direct smears of the effusion showed only occasional eosinophiles, scattered leukocytes and no bacteria.

Suehs[25] saw only one eosinophile in smears obtained from some 50 patients. Robison and Nicholas[26] found no eosinophiles in a hundred subjects with acute and chronic otitis media and secretory catarrh. In some 50 cases of serous otitis studied by the Bryans[27] all smears were negative for eosinophiles.

As part of a comprehensive study of tubo-tympanitis, Carr and Senturia[28] examined the histories and aspirated effusions obtained from 151 ears (107 patients). Despite a thorough cytologic study using Wright's and Papanicolaou stains, only six of the smears showed a single eosinophile on the entire slide; one specimen showed five cells per high power field.

A short time ago we presented our evidence in support of the view that the largest percentage of middle ear effusions

are not allergic but inflammatory in origin.[29] A number of the specimens revealed a thin, low viscosity secretion in which there was an absence of inflammatory cells (serous type). Some of the specimens were typical of suppurative otitis media and presented classical inflammatory cytologic findings (purulent type). The largest number had microscopic characteristics of a subacute or chronic inflammation (mucopurulent).

A very small number of our specimens showed a large amount of mucoid material and no bacteriologic or cellular elements. The findings suggested that there had been an out-

TABLE I.

		Characteristics		
Diagnosis	Synonyms	Cellular Remnants	Neutrophiles	Mucous Strands
Serous	Otitis media with effusion Secretory otitis Secretory catarrh	Rare	Rare	None
Purulent	Suppurative otitis media Acute otitis media	Moderate	Numerous	Rare
Mucopurulent	Chronic otitis media Glue ear Catarrhal otitis media Mucoid ear	Numerous	Few	Numerous
Mucoid	Allergic otitis media (?) Glue ear	Rare	None	Myriads

pouring of sterile mucoid secretions without any classical inflammatory components. It is conceivable that these may constitute the allergic group, notwithstanding our inability to demonstrate the presence of eosinophiles (see Table I).

The following working classification of middle ear effusions was set up on the basis of the available literature and our own clinical and cytologic findings.[28]

In an effort to understand the pathogenesis of these conditions, effusions were produced experimentally in dogs and were studied in the same manner as in humans.[30] It is our

impression that the data provides additional support for the earlier conclusion that most effusions in humans are of inflammatory origin.

I am in agreement that many patients with middle ear effusions show an allergic diathesis, including a family or personal history of allergy, pale, hypertrophic nasal mucous membrane, mucopurulent secretion in the nasal fossae with increased numbers of eosinophiles and lymphoid hyperplasia of the naso- and oropharynx. These individuals appear to be more susceptible to upper respiratory infections and possibly are more prone to middle ear effusions.

From what has been said above, it is clear that there is an imposing lack of evidence to substantiate the claim of an allergic etiology for the great majority of cases of otitis media with effusions; therefore, it is suggested that immediate treatment should be directed to the control of the inflammatory process in the middle ear and sinuses and, following this, attention should be given to the discovery and elimination of the factors which are contributing to the allergic state.

SUMMARY.

As indicated at the beginning of this presentation, it is my feeling that there is a distinct place for the diagnosis of allergic otitis externa and otitis media. There is some clinical evidence that these conditions constitute a consistent though small percentage of our cases in otologic practice. They are frequently misdiagnosed and, therefore, respond poorly to the conventional therapeutic approach.

I am hopeful that a greater appreciation and understanding of the allergic dermatoses will permit earlier diagnosis and therapy and, therefore, will effectively circumvent the aggravating superimposed acute dermatitis.

More investigative work is necessary to delineate adequately the allergic middle ear since we have few reliable yardsticks to differentiate clearly these allergic effusions from inflamma-

tory conditions in the tympanum. We are confident that progress is being made in understanding the allergic otitides.

The author is indebted to Dr. Carl Gessert, Charles Carr, Elizabeth Baumann and Dorothy Baldwin for assistance in the preparation of this manuscript, and to Dr. M. D. Marcus for suggestions and criticisms.

REFERENCES.

1. Senturia, B. H.: "Diseases of the External Ear." Charles C. Thomas Co., Springfield, 1957.

2. Hoople, G. D.: Otitis Media with Effusion. *Trans. Amer. Acad. Ophthal. and Otolaryn.*, 54:531, May-June, 1950.

3. Koch, H.: Allergical Investigations of Chronic Otitis. *Acta Otolaryngol.*, Suppl. 62:1-201, 1947.

4. Derlacki, E. L.: Aural Manifestations of Allergy. *Ann. Otol., Rhinol. and Laryngol.*, 61:179-188, 1952.

5. Kuhn, H. A.: Aural Allergy. *Trans. Amer. Acad. Ophthal. and Otolaryn.*, pp. 201-205, March-April, 1952.

6. Williams, H. L.: "Meniere's Disease." Charles C. Thomas, Springfield, 1952.

7. Rostenberg, A., Jr.: Cutaneous Allergic Disorders; a Review of Fundamental Theory with a Discussion of Certain Clinical Entities. *Med. Clin. No. Amer.*, 33:177-204, 1949.

8. Rackemann, F. M.: History Taking in Allergic Disease. *Jour. A.M.A.*, 106:976-979, 1936.

9. Steele, J. M.: Diagnosis of the Allergic State. A Point Scoring System. *Ann. Allergy*, 14:1-7, 1956.

10. Senturia, B. H., and Marcus, M. D.: Etiologic Classification of Diseases Involving the External Ear. *Ann. Otol., Rhinol. and Laryngol.*, 61:18-32, 1952.

11. Sophian, L. H., and Senturia, B. H.: The Pathology of Acute Diffuse External Otitis. *Ann. Otol., Rhinol. and Laryngol.*, 64:300-318, 1955.

12. Sophian, L. H.; Cooper, Z. K. and Senturia, B. H.: Pathologic Changes of the Skin of the External Auditory Canal in Chronic Otitis Media and Mastoiditis. *Ann. Otol., Rhinol. and Laryngol.*, 63:261-272, 1954.

13. Jones, E. H., and Norman, T. D.: Preliminary Report on the Pathogenesis of Dermatoses of the External Auditory Canal: Twenty Cases of Acute Diffuse External Otitis, Due to Deep Infection. The Laryngoscope, 68:839-844, 1958.

14. Senturia, B. H., and Carr, C. D.: Studies of Factors Considered Responsible for Diseases of the External Ear. I. Survival of Exogenous Bacteria in the External Auditory Canal Under Varied Conditions. The Laryngoscope, 68:2052-2079, 1958.

15. Dohlman, G.: Allergiska Processer i Mellanörat. *Nord. Med. Tidskr.*, 17:224-226, 1943.

16. Dohlman, G.: Till Fragen om den Kroniska Otitens Behandling. *Nord. Med. Tidskr.*, 20:2236-2238, 1943.

17. Derlacki, E. L.: The Middle Ear. *Trans. Amer. Acad. Ophthal. and Otolaryng.*, pp. 91-96, Jan.-Feb., 1957.

18. Dean, L. W., Jr., and Pfingsten, M. G.: A Chemical and Cytologic Study of Aural and Nasal Exudates as Regards the Chemical Diagnosis of Cholesteatoma. *Ann. Otol., Rhinol. and Laryngol.*, 42:484-496, 1933.

19. Ojala, L., and Palva, T.: Macrophages in Aural Secretions and Their Clinical Significance. The Laryngoscope, 65:670-692, 1955.

20. Lewis, E. R.: Otitis Media and Allergy. *Ann. Otol., Rhinol. and Laryngol.*, 38:185-188, 1929.

21. Proetz, A. W.: Allergy in the Middle and the Internal Ear. *Ann. Otol., Rhinol. and Laryngol.*, 40:67-76, 1931.

22. King, J. T.: The Condition of Fluid in the Middle Ear: Factors Influencing the Prognosis in 56 Children. *Ann. Otol., Rhinol. and Laryngol.*, 62:496-506, 1953.

23. Ivstam, B.: On Secretory Catarrh of the Middle Ear with Special Reference to its Pathogenesis and Prognosis. *Acta Oto-Laryngol.*, 3:274-285, Suppl. 116, 1954.

24. Jordan, R.: Chronic Secretory Otitis Media. The Laryngoscope, 59:1002-1015, 1949.

25. Suehs, O. W.: Secretory Otitis Media. The Laryngoscope, 62:998-1027, 1952.

26. Robison, J. M., and Nicholas, H. O.: Catarrhal Otitis Media with Effusion. *South. Med. Jour.*, 44:777-789, 1951.

27. Bryan, W. T. K., and Bryan, M. P.: Personal communication.

28. Carr, C. D., and Senturia, B. H.: Cytology of Middle Ear Effusions. Unpublished data.

29. Senturia, B. H.; Gessert, C. F.; Carr, C. D., and Baumann, E. S.: Studies Concerned with Tubo-tympanitis. *Ann. Otol., Rhinol. and Laryngol.*, 67:440-467, 1958.

30. Sade, J.; Carr, C. D., and Senturia, B. H.: Middle Ear Effusions Produced Experimentally in Dogs. I. Microscopic and Bacteriologic Findings. To be published.

OTOLOGICAL FUNCTIONAL MICRO-SURGERY POST GRADUATE COURSE.

A practical course of surgical techniques in otological surgery will be conducted by Dr. Michel Portmann at the University of Bordeaux, France, Ear, Nose and Throat Department, June 26 to July 5, 1960. This course will be given in English.

The number of assistants will be limited to twenty, divided into two groups. The fee of $100.00 is to be paid at the Faculty of Medicine of Bordeaux, at the beginning of the course. Applications should be sent to Dr. Michel Portmann, 45 Cours Foch, Bordeaux, France.

TUMORS OF THE FRONTAL SINUS.*

DELMAR F. WEAVER, M.D.,

Detroit, Mich.

A survey of the medical literature dealing with tumors originating in the frontal sinus and those of the surrounding areas indicates that a number of competent observers have written articles on the subject down through the years. Since many of these tumors rarely occur, often the article follows a general pattern. First there is a review of the literature on the particular tumor followed by statistics regarding the relative frequency of occurrence; the total number of cases reported to date; the various theories as to their origin and helps in diagnosis and methods of treatment. Usually there are reports of several cases from the authors' personal experience. Each article adds to the total store of information and is of immeasurable value towards a complete understanding of the subject; however, it may be worth while to approach the subject from an overall standpoint in an effort to facilitate the evaluation and handling of patients who present themselves with evidence of such tumors.

The frontal sinus is a mucous membrane lined space which connects the orbit, nasal cavity, brain and brow. It appears to be of no use to modern man. Only a few types of tumors originate in it, and each of these is seen infrequently. Tumors originating in the surrounding structures are relatively uncommon, and invasion of the frontal sinus by these is seen infrequently also; however, the presence of a tumor in this general area may portend a very grave problem for any particular patient. Not only is visual acuity apt to be affected when the orbit is involved but erosion of the bone over the dura mater also may lead to dangerous and fatal intracranial complications. Consequently, it is essential that there be reciprocal understanding between the representatives of the

*Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Inc., Chicago, Ill., Jan. 18-19, 1960.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 20, 1960.

several specialized fields concerned so that the patient may benefit from an organized attack on the problem.

Following a survey of most of the tumors that may occur in the area under discussion, with examples of some of them, the case history of a patient will be given in some detail. This case demonstrates the very real problem of diagnosis that may exist in certain cases even in the hands of competent observers.

Primary tumors of the frontal sinus may be benign or malignant. The benign tumors consist of osteomas, mucoceles and cystic tumors resembling mucoceles, including hemorrhagic cysts and cholesteatomas.

In 1938 Hempstead[1] wrote an excellent article on osteomas. The literature in more recent years has included articles by Brunner and Spiesman,[2] Gibson and Walker,[3] Zovickian and Cooper,[4] Vadala and Somers.[5] Several of these referred to an earlier article by Teed.[6]

Osteomas may be composed of eburnated or cancellous bone or a mixture of the two. They may be attached to the interior of the frontal sinus at any point, but the attachment is usually by cancellous bone. Small osteomas not obstructing the drainage of the sinus are usually discovered only on roentgenological examination. The density of the shadow is that of bone. Further roentgenological examination of these tumors should be made from time to time, and if there is evidence of enlargement, removal should be advised. This should be possible through the floor of the frontal sinus. If small tumors obstruct drainage, symptoms of the obstruction may be present, or a mucocele may be formed requiring operation.

If they are first seen at a stage when erosion through the floor, intersinus septum or anterior or posterior table may have taken place, there may be evidence of involvement of the orbit, the other sinus, brow or meninges. Expansion toward the brain may result in meningitis, brain abscess, pneumatocele or cerebrospinal rhinorrhea, and there may be deep involvement of the brain substance. Removal of the larger tumors requires wider exposure. It may be necessary to

remove the anterior table without conserving the supraorbital ridge. Zovickian and Cooper[4] have used cancellous bone chips from the iliac crest to repair the defect resulting from the removal of a large osteoma from the frontal sinus.

Brunner and Spiesman[2] feel that all osteomas of the frontal sinus can be removed by the external frontal approach. Hempstead,[1] however, stated that while most of them can be removed through the fronto-ethmoid incision, if there is evidence of involvement of the dura mater sufficient to indicate the likelihood of a tear or if other intracranial complications are present, the transfrontal approach from the dural side is best.

The mucocele is a tumor which has some of the characteristics of an osteoma. Kaplan, Schwartz and Metson[7] have discussed various of its aspects. It consists of a closed cavity lined with mucous membrane which continues to secrete mucus and to fill the cavity with these secretions. It begins as a mechanical block to the drainage of the sinus itself or to the glands of its lining mucosa. The mucosa of the sinus continues to produce its normal mucoid secretion, but lacking normal outlet, the secretion accumulates and fills the sinus. Continued expansion may cause erosion of the sinus wall and encroachment on any of the adjacent cavities. If into the nose, air may pass along the sides and result in a large extra or intradural pneumatocele. The cavity most commonly involved secondarily is the orbit and, for this reason, the patient is generally first seen by the ophthalmologist. With dural involvement the intracranial complications may be the same as those associated with osteoma.

When the contents of a mucocele are invaded by bacterial infection a pyocele is formed.

On roentgenological examination the mucocele is represented by a fluid or soft tissue density which may lessen as bony erosion progresses. The presence of a cystic tumor in the superior or supermedial quadrant of the orbit should incite one's suspicion of mucocele. Primary orbital tumors are more likely to involve the lateral quadrants. If there is evidence of a mucocele of the frontal sinus, exploration through a

fronto-ethmoid incision is indicated and if present, it should be removed in its entirety.

A variety of suggestions regarding the nasofrontal duct has been made by many authors; however, since this is an individualistic problem and more pertinent to chronic infections of the sinus, no attempt will be made to evaluate it at this time. There is some reason to believe that frontal duct problems may be less likely to arise after mucocele removal than following operation for chronic infections of the frontal sinus.

Simonton and Medwick[8] have discussed tumors of the frontal sinus simulating mucocele, including hemorrhagic cysts and cholesteatomas.

Primary malignant tumors of the frontal sinus are quite rare. Sciarra and Hallberg[9] reported one case of squamous cell carcinoma, grade III, which arose in the frontal sinus without question. It had eroded both tables of the frontal sinus, and the dura was involved. The patient was alive and well for at least 12 years after simple excision and implantation of radium. They found a total of 90 cases reported as primary malignant tumors of the frontal sinus. This included their own case; however, the possibility of some duplication of cases was raised and also a question of whether or not all of them actually originated in the frontal sinus, since in some cases the other sinuses also were involved.

The term secondary tumors is used to denote those that have invaded the frontal sinus from a primary origin in surrounding areas and includes those tumors of the skin, subcutaneous tissue and muscle which lie in such close proximity to the sinus that it is thought inadvisable to exclude them. This group includes nevi, dermoid, epidermoid and sebaceous cysts, fibromas, lipomas, fibrolipomas, neurofibromas, hemangiomas and certain inflammatory tumors as tuberculomas and Boeck's sarcoid. When small enough these tumors can be removed in one stage by simple excision. Certain superficial skin tumors such as pigmented nevi may be so large that they must be removed in stages several months apart to prevent distortion of the lids or brow. Cavernous

hemangiomas in this location may be best handled by the injection of sclerosing material or subcutaneous electrocoagulation with an electrode insulated except for its tip.

Malignant tumors of the overlying skin which invade the frontal sinus consist chiefly of basal or squamous cell epitheliomas or a mixture of the two. I have never seen a melanoepithelioma in this location but presume it could occur. When these tumors have not advanced beyond the subcutaneous tissue and muscle and the periosteum has not been invaded, the chance of curing them by excision is above 90 per cent. The margin of normal tissue removed should be as wide as feasible; however, if the periosteum has been invaded as indicated by fixation of the tumor or the tumor has been previously irradiated and recurred or if it is infected following inadequate removal, the chance of a cure is much less.

In these cases more extensive surgery in indicated and should be planned with the eradication of the tumor the primary objective. Repair of the postoperative defect can be carried out later. While cold instrument removal of the tumor and the surrounding soft tissue and bone may be suitable in some cases, more often destruction by electrocoagulation is best. It eliminates the chance of grafting free tumor cells onto normal tissue and will probably destroy a larger area of tumor with less postoperative defect; however, there is no limit to the extent of removal of tissue that can be carried out with this method. When bone has been electrocoagulated to any extent, it will be necessary to remove a sequestrum after about two months.

I have operated on six patients who had basal or squamous cell epitheliomas which had originated in the skin around the frontal sinus and had invaded it when first seen by me. All had had a variety of combinations of treatment which included cold knife excision, electrocoagulation and irradiation with both radium and X-ray. Such treatment had been carried out on several different occasions by several different clinicians.

Our treatment was thorough electrocoagulation in five cases and cold instrument excision in one case. This patient's tumor

was attached to the floor of the frontal sinus, and it was removed along with the floor of the frontal sinus. Plastic closure of the wound was carried out immediately.

Four of the patients are alive and without evidence of recurrence 18, 14, 12 and two-and-a-half years since operation.

A fifth patient, an 83-year-old man, had been free of recurrence for 12 years when he died soon after an accident. This accident occurred in June, 1959 while escaping from a hotel fire in Stalheim, Norway.

The sixth patient is under observation now, having been operated on first in September, 1959.

Both tables of the frontal sinus exposing the dura widely were removed from one patient. The outer table of the frontal sinus with the associated membrane was removed from two patients. This exposed the sinus cavity.

The outer table of the frontal sinus without perforation of the lining membrane was removed from two patients, and only the floor was removed from the patient who had cold instrument surgery as mentioned above.

Five of the postoperative defects were covered with contiguous pedicled flaps. Three of the flaps had a single pedicle, and a free graft was used to cover the donor area. Two of the flaps were bi-pedicled, and no free graft was used. Three of these patients have been mentioned in more detail in other publications.[10,11]

Benign tumors such as osteomas and cysts originating in the ethmoid sinus and invading the frontal sinus may produce a frontal duct problem. In small frontal sinuses this problem may be resolved in certain cases by removing the entire frontal sinus floor enabling the orbital contents to obliterate it. All of the lining membrane must be removed in such cases.

While malignant tumors arising in the ethmoid sinus region which invade the frontal sinus are usually squamous cell carcinomas, adenocarcinomas and sarcomas do occur. The removal of them whether by cold instrument surgery, electro-

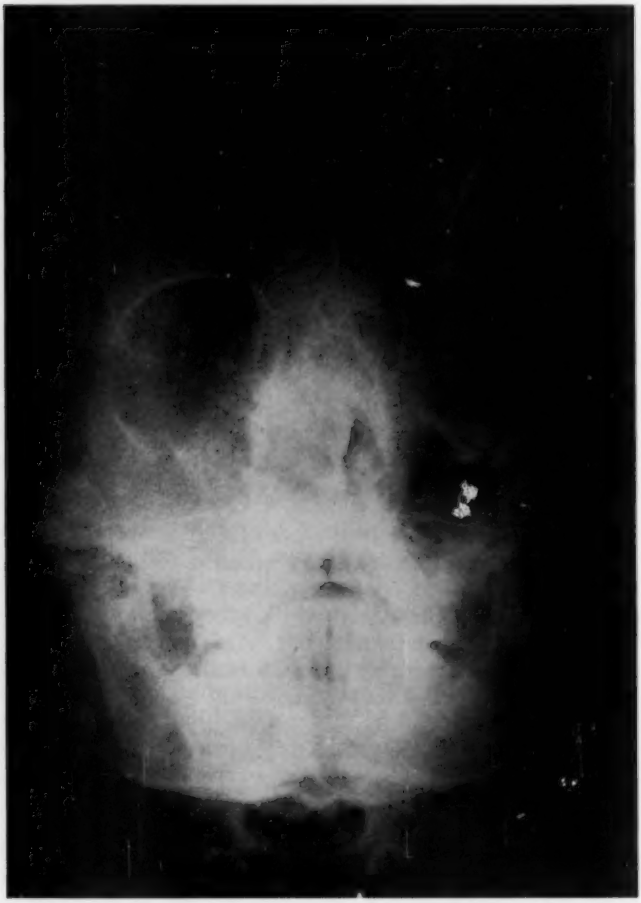


Fig. 1. Chondrosarcoma involving both frontal and ethmoid sinuses, nasal cavity and right maxillary sinus region.

coagulation or irradiation is simply an extension of the treatment from the primary location.

At times it may be necessary to extend surgical treatment into the frontal sinus to provide drainage even though it is not involved by tumor.

Fig. 2. Chondrosarcoma involving skull in region of cribriform plate of ethmoid bone.

A 14-year-old boy was sent to me for a tonsillectomy and adenoidectomy in October, 1959. My attention was called to a tumor in the right nasal cavity. It was quite firm. After slight manipulation it was necessary to pack the nose tightly to stop profuse hemorrhage. Roentgenograms of the sinuses showed extensive involvement of the nasal cavity, both ethmoid and frontal sinuses and the right maxillary sinus region (see Figs. 1 and 2). Biopsy was reported as chondrosarcoma.

On October 15, 1959, under pentothal sodium anesthesia with an intratracheal tube in place, the tumor in the right nasal cavity was electrocoagulated as thoroughly as possible and then through a fronto-ethmoid incision extensive electrocoagulation was carried out to provide adequate drainage. This extended through the nasal septum into the opposite

Fig. 3. Mucocele of left frontal sinus.

ethmoid region. The tumor appeared to involve the skull in the region of the cribriform plate. The right nasal bone and nasal process of the superior maxilla had been destroyed by the tumor. The floor of the right frontal sinus was removed and the sinus found to be filled by the tumor. Six capsules containing 10 milligrams of radium each were distributed throughout the cavity and left in place for 46 hours (2760 mgm. hours). His condition following operation appears to be improved.

Fig. 4. Complete erosion of supraorbital ridge by large mucocele.

Little encouragement has been given his parents regarding the final outcome.

While tumors originating in the orbit are not common in the practice of the average ophthalmologist there are publications which analyze rather large groups of these tumors.

One such report was published by Benedict[12] in 1949 and was based on a study of 740 orbital tumors. There were 33 pathological types diagnosed, and 420 of them were operated on.

These tumors produce exophthalmos, but the amount of proptosis of the globe and direction of its displacement only in general indicate the size and position of the tumor within the orbit. Exophthalmic goiter may produce unilateral exophthalmos and certain inflammatory diseases may do so also.

Fig. 5. Posterior table of frontal sinus intact in 1956.

A list of orbital tumors includes hemangiomas, which constitute about a third of them, endotheliomas, osteomas, meningiomas, dermoid cysts, melanomas and mixed tumors and cylindromas (in the lacrimal gland). Metastatic carcinoma of the breast, stomach and intestine may involve the orbit. It is seldom that one can make a diagnosis of any particular type of orbital tumor from clinical evidence alone. They may be confused with mucocele or pyocele of the frontal sinus.

Progressive loss of visual acuity may be the outstanding sign of orbital tumors and is a definite indication for operation.

Three-hundred-and-forty of Benedict's 420 operated tumors were removed by the frontal or fronto-lateral approach. This approach is usually proper for tumors of the anterior third of the orbit as well as those that lie along the superior wall.

Fig. 6. Marked erosion of posterior table of frontal sinus in 1958.

Resection of the lateral wall of the orbit (Kronlein's operation) is most suitable for orbits with small outlets and for certain tumors situated in the posterior third of the orbit. Other tumors situated in the posterior third and especially those that may be present in both the orbit and cranial cavity, as endotheliomas and hemangiomas, can be reached more directly through a transcranial approach.

While, theoretically, many orbital tumors may involve the

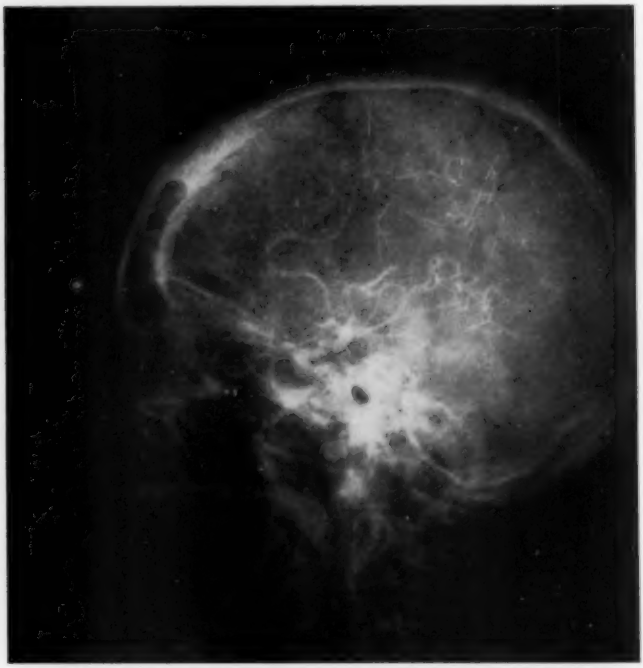


Fig. 7. Arteriogram showing moderate posterior displacement of anterior cerebral artery.

frontal sinus, and we may be called in consultation on these cases, our chief interest in them concerns the problem of diagnosis. In general, those tumors which can be palpated in the frontonasal or medial quadrants of the orbit would tend to be associated with the frontal or ethmoid sinus. These tumors can be approached through the fronto-ethmoid incision. Tumors that lie in a more lateral position, especially if roentgenograms of the sinuses are negative, are apt to be in the realm of ophthalmology.

The meningioma is about the only intracranial tumor that may invade the frontal sinus often enough to merit consideration. This occurrence is quite rare since they usually arise more posteriorly and seldom reach so far forward.

CASE REPORT.

A 73-year-old white woman was first seen by me on November 29, 1958.

She stated that four or five years previously she developed a slight swelling over the left eye associated with periodic closure of the left eyelid due to periodic increase in the swelling. The permanent swelling had gradually increased until the present time.

She had a history of moderately advanced generalized Paget's disease, moderately severe diabetes with far advanced vascular disease and arteriosclerotic and hypertensive heart disease. There was a history of having had a coronary thrombosis, and she had been in congestive failure. She was taking Quinidine for paroxysmal tachycardia.

She had been seen alternately by two excellent ophthalmologists over a period of years. These men were not associated. Their reports are as follows:

"There was no swelling of the orbit in May, 1952. In 1955 vision was 20/20 in both eyes and no tumor or swelling was present. There was some retinal hemorrhage at that time, however. On October 30, 1956 there was invasion of the orbit by tumor and blurring of the nasal margin of the nerve head with increased pressure. In April, 1957 this tumor was palpable at the orbital rim nasally, above and below. Her vision was still good so, in view of the presence of the general diseases, surgery was not advised at that time. In January, 1958 there was a massive tumor in the supraorbital region depressing the eye. Retinal hemorrhages were present. Surgery was urged at that time."

In March, 1958 she was complaining bitterly of pressure and discomfort. Neurological consultation was obtained at this time, and arteriograms were made. The patient was boarded for intracranial surgery but it was decided against due to the apparent likelihood of hopeless intracranial involvement. Her internist considered it likely that the tumor was from sarcomatous degeneration of the frontal bone since sarcomas develop in a significant number of cases of Paget's disease. He felt that removal of the tumor with the eye was justified in view of the discomfort she was having. He referred her to a third ophthalmologist in May, 1958. He aspirated the tumor, found that it contained fluid and referred her to me. At this time visual acuity was very poor in the affected eye.

On examination a large, firm, tense tumor was evident in the superior half of the left orbit. This markedly displaced the globe downward. It was difficult to separate the eyelids (see Fig. 3).

Roentgenograms showed the tumor extensively involving a large frontal sinus with complete erosion of the supraorbital ridge (see Fig. 4). Lateral films made in 1956 showed the posterior table of the frontal sinus intact (see Fig. 5), while recent films showed it markedly eroded (see Fig. 6). Arteriograms showed moderate posterior displacement of the anterior cerebral artery (see Fig. 7).

On December 4, 1958, under pentothal sodium anesthesia with an intratracheal tube in place, a fronto-ethmoid incision was made which extended to the outer extremity of the eyebrow. Almost immediately a large mucocele was exposed which contained at least four ounces of thick liquid of a light green color. It did not appear to have been invaded by infection to a significant degree. The floor of the frontal sinus was completely absent due to erosion of the cyst, and this was also true of the supraorbital ridge. The posterior table of the frontal sinus was likewise eroded exposing the dura over a circular area 4 cm. in diameter. All of the cyst wall was removed, and the frontonasal duct was recon-

Fig. 8. Appearance five weeks after operation.

structed. A size 26 F rubber catheter was placed through the nose and frontal duct into the frontal sinus cavity; however, hemorrhage was so troublesome that it was necessary to remove the tube and pack the nasal cavity with Iodoform gauze. This was removed in about a week. The postoperative course was uneventful except for a moderate mental depression which disappeared in a few days. The temperature did not become elevated above normal during the nine days she was in the

hospital. The eye soon tended to return to a more normal position, and there was some improvement in vision (see Fig. 8).

The pathological report was acute and chronic inflammation of the frontal sinus.

Three months after operation the patient developed slight swelling of the left lower eyelid. A large curved suction tip was passed into the sinus through the frontal duct dilating the duct sufficiently so that the swelling soon subsided. Several months later she developed swelling but was seen by an otolaryngologist near her home who drained the sinus externally. A recent report from him states that she is having no trouble now.

An attempt has been made to present a bird's-eye view of tumors of the frontal sinus in an effort to facilitate the handling of patients with evidence of these tumors.

The case history of a patient with a large mucocele of the frontal sinus has been presented in some detail. This history demonstrates the extreme confusion that may be associated with the diagnosis of tumors that arise in the frontal sinus, even when observed by thoroughly competent men in fields closely allied to otolaryngology.

One is tempted to suppose that inadequate drainage of the frontal sinus resulting from the deformity of Paget's disease was responsoble for the formation of the mucocele.

BIBLIOGRAPHY.

1. Hempstead, Bert E.: Osteomas of the Paranasal Sinuses and the Mastoid Process. Report of Cases. *Jour. A.M.A.*, 111:1273-1275, Oct. 1, 1938.

2. Brunner, H., and Spiesman, I. G.: Osteoma of the Frontal and Ethmoid Sinuses. *Ann. Otol., Rhinol., and Laryngol.*, 57:714-737, Sept., 1948.

3. Gibson, T., and Walker, F. M.: Large Osteomas of the Frontal Sinus. *Brit. Jour. Plast. Surg.*, 4:210-217, Oct., 1951.

4. Zovickian, Anthony, and Cooper, Philip: Osteoma of the Frontal Sinus: Removal Combined with Immediate Repair of the Frontal Bone Defect. *Plast. and Reconst. Surg.*, 19:150-155, Feb., 1957.

5. Vadala, A. J., and Somers, Lt. Col. Kenneth: Osteoma of the Frontal Sinus. *Arch. Otolaryngol.*, 50:618, Nov., 1949.

6. Teed, W. A.: Primary Osteoma of the Frontal Sinus. *Arch. Otolaryngol.*, 33:255-292, Feb., 1941.

7. Kaplan, Samuel; Schwartz, Abiah, and Metson, Bates: Mucocele of the Frontal and Ethmoid Sinuses. *Arch. Otolaryngol.*, 51:172-187, Feb., 1950.

8. Simonton, Kinsey, and Medwick, Joseph: Epidermoid Like Lesions of the Frontal Bone Simulating Mucocele. Report Two Cases. *Ann. Otol., Rhinol. and Laryngol.*, 57:473-482, June, 1948.

9. SCIARRA, PASCHAL A., and HALLBERG, O. ERIK: Primary Squamous Cell Carcinoma of the Frontal Sinus. *Ann. Otol., Rhinol. and Laryngol.*, 65:225-229, March, 1956.

10. WEAVER, D. F.: Free Skin Grafts and Pedicle Flaps in the Treatment of Recurring Basal Cell Epitheliomas. Case Reports. THE LARYNGOSCOPE, 53:336-342, May, 1942.

11. WEAVER, D. F.: Tumors of the Face. *Arch. Otolaryngol.*, 52:234-252, Aug., 1960.

12. BENEDICT, WILLIAM L.: Surgical Treatment of Tumors and Cysts of the Orbit. *Amer. Jour. Ophthalmol.*, Vol. 32, No. 6, Pt. 1, June, 1949.

THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The program of the West Virginia Academy of Ophthalmology and Otolaryngology has now been completed for their annual meeting at the Greenbrier Hotel, White Sulphur Springs, W. Va., April 10-12, 1960. Guests speakers will be: Dr. Harold G. Scheie, Professor of Ophthalmology, University of Pennsylvania School of Medicine, Philadelphia, Pa., "The Surgical Management of Glaucoma," Part I and Part II; Dr. Charles E. Iliff,Associate Professor of Ophthalmology, John Hopkins University School of Medicine, Baltimore, Md., "Adult Ocular Tumors"; Dr. Oscar T. Becker, Clinical Associate Professor of Otolaryngology, University of Illinois School of Medicine, Chicago, Ill., "Plastic Surgery in Otolaryngology"; Dr. Helen J. Ossofsky, Assistant Professor of Pediatrics, Georgetown University, Washington, D. C., "Pediatric Ocular Tumors"; Dr. John T. Dickinson, Department of Otology, University of Pittsburgh, Pittsburgh, Pa., "Modern Treatment of Otosclerosis."

There is a non-member registration fee of $10.00 to cover all the social and scientific sessions. For further information contact the secretary, Dr. A. C. Esposito, First Huntington National Bank Building, Huntington, W. Va.

LARYNGEAL STENOSIS.*

MERCER G. LYNCH, M.D.,
and
FRANCIS E. LEJEUNE, M.D.,

New Orleans, La.

We herewith present a method for the correction of laryngeal stenosis produced by web formation between the cords.

Laryngeal stenosis as the result of infectious, inflammatory reactions, such as diphtheria, tuberculosis, syphilis, typhoid fever, erysipelas, and smallpox is seen only in exceedingly rare instances now. Today trauma to the larynx from transportation accidents, war wounds, surgical injury, and irradiation is much more likely to be the cause of laryngeal stenosis. Trauma to the larynx from transportation accidents, war wounds and irradiation usually produces a form of stenosis with involvement of the entire larynx. These cases are dealt with in an entirely different manner. I will stress mainly those cases of congenital web formation or those following surgical injury. As a general rule, congenital web formation involving the larynx occurs from the formation of a web between the vocal cords. Usually, this web is very thin or moderately thick; seldom is it ever very dense fibrous tissue and it may be corrected by the method to be mentioned.

Surgical injury to the anterior commissure is usually the result of over-enthusiasm on the part of the surgeon. In the removal of multiple papillomas of the vocal cord or of tumors involving the anterior commissure, the virginity of the commissure is violated, and there is a raw surface involving both cords in the anterior third. As a result of this, scar tissue develops with formation of a web. This web is usually moderately thick, but involves only the commissure itself, extending posteriorly on each side, depending upon the amount of injury and raw surface produced by removal of the growth

*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., New Orleans, La., Jan. 15, 1960.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 22, 1960.

or unnecessary trauma to the cords; however, the web itself is limited entirely to the cords. If this web is extremely thick and fibrous, it must be approached from an external laryngofissure incision, with removal of the web and insertion of a tantalum splint, to prevent approximation of the raw surfaces of the cords; or, it may be removed and some form of laryngeal stent inserted between the cords in the form of a plastic mold, a hard rubber mold, or a metal mold. This, however, necessitates tracheotomy for a period of time. In either of these procedures, an external incision is necessary. In an attempt to overcome the necessity for external surgical procedure we have tried several methods. We present the latest procedure. This method, however, can be used only in those patients in whom the web itself is thin or moderately thick; in a dense fibrous scar it is not practical.

The patient is suspended, using the suspension laryngoscope. In this way, one is able to use both hands to perform the operation. After adequate exposure of the larynx a small opening is created as close to the anterior commissure between the vocal cords as is feasible. A small polyethylene tube, through which heavy black silk thread or silver wire has been passed, is then inserted through the opening created. The polyethylene tube and silver wire now extend subglottically as well as supraglottically. A Riverden needle is introduced externally in the medium line of the neck, through the cricothyroid membrane into the subglottic portion of the larynx; the silver wire is fed into the slot of the Riverden needle and the needle is then withdrawn externally bringing with it the silver wire. This same procedure is used by inserting the Riverden needle through the thyro-hyoid membrane. The position of the polyethylene tubing is carefully checked, and if found to be satisfactory, the silver wires are anchored externally by appropriate metal buttons and lead shot. The polyethylene tube is left in place for a period of six weeks to two months. There is no interference with respiration, tracheotomy is not necessary and as a rule the tube is well tolerated. The patient is examined at intervals, and when epithelization has occurred, the larynx is again exposed with the aid of the suspension laryngoscope. The tube is grasped from within

the larynx, the wires are cut externally and the tube is removed. The web is still present and attached to both vocal cords, but there is now an epithelized opening at the anterior commissure. The web is excised along the edges of the cord, care being taken not to injure the true cord. There is no raw surface at the anterior commissure; hence, the web does not recur.

We are indebted to Justo Alonso for first describing use of the Riverden needle in the insertion of an acrylic mold in the larynx for correction of stenosis and webs. Since then we have used the polyethylene tube with excellent results in a limited number of cases with either thin or moderately thick web formation between the cords.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 34th convention of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Illinois Hotel in Bloomington, Ill., on April 22nd, 23rd and 24th.

Our guest speakers are: Dr. John W. Henderson, Rochester, Minn., Ophthalmology; Dr. Z. Z. Godlowski, Chicago, Ill., Otolaryngology. Member participants are Dr. W. A. McNichols, Dixon, Ill., Ophthalmology; Dr. George H. Woodruff, Joliet, Ill., Otolaryngology.

Prof. Arthur W. Watterson, of the Geography Department of the Illinois State Normal School, Bloomington, Ill., will be the speaker at the Saturday evening banquet.

For further information please address Dr. C. L. Pannabecker, Secretary-Treasurer, 434 Jefferson Bldg., Peoria, Ill.

SEQUELAE OF TRACHEOBRONCHIAL ASPIRATION.

CARL J. BRIDGE, M.D.,

Gardner, Mass.

A small series of cases is presented for demonstration of short and long range pathologic changes associated with suctioning of the tracheobronchial tree through a tracheotomy stoma.

Tracheotomy is performed if the upper respiratory tract is a primary source of obstruction; if the patient is unable to aid at all in removal of secretions and exudates, or if suction will be required over a long period of time. It is obvious that the extent to which suction is required is dependent upon the patient's ability to cough. When the cough reflex is depressed, the introduction of a catheter or bronchoscopy tube aids in its stimulation, and secretions in the lower portions of the respiratory tract can be expelled, providing there is some air in the alveoli distal to them.

Blind suction of the trachea and primary bronchi is thought to be almost as effective as direct vision suctioning through a bronchoscope, when liquid secretions or exudates are involved. Blind suctioning is also believed to be relatively atraumatic when done with a No. 16 French soft rubber catheter with a hole in the end and one in the side, just proximal to the end, under a negative pressure of from five to ten pounds. If less than five pounds, it is ineffective; if more than ten pounds, the catheter collapses, thus preventing the bronchial and tracheal mucosa from becoming unduly traumatized. At times a curved tipped catheter with a hole in the end has been used, in the belief that it aids in the removal of material from secondary bronchi. When there are indications that blind suction is inadequate for removal of material from the tracheobronchial tree, we resort to direct vision bronchoscopy.

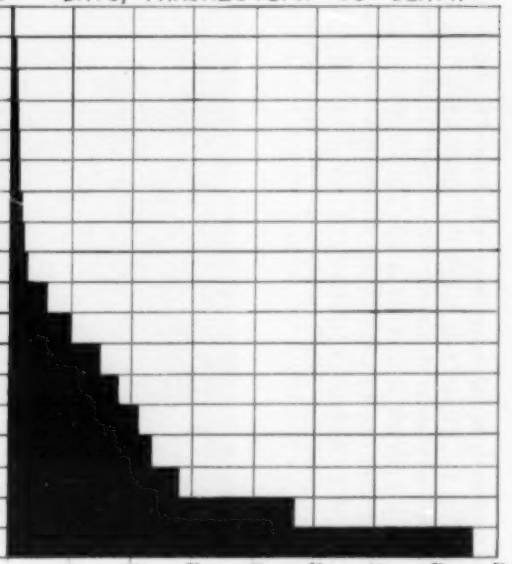
A series of 18 cases of poliomyelitis, from the Boston epidemic of 1955, treated at the Massachusetts General Hospital,

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 20, 1960.

FIGURE 1.

EFFECTS OF TRACHEOBRONCHIAL ASPIRATION IN FATAL POLIOMYELITIS CASES

	PULMONARY DIAGNOSIS	TRACHEA & BRONCHI GROSS	TRACHEA & BRONCHI MICROSCOPIC	AGE	DAYS, TRACHEOTOMY TO DEATH
1.	EDEMA, SEVERE	FROTHY FLUID, COPIOUS	CONGESTION AND EDEMA, WALL	31	
2.	EDEMA	HEMORRHAGIC MUCUS, FOAM		21	
3.	CONGESTION, EDEMA, BASAL ATELECTASIS	SERO-SANGUINOUS EXUDATE		35	
4.	CONGESTION, EDEMA, ATELECTASIS	MUCO-HEMORRHAGIC EXUDATE		50	
5.	CONGESTION, EDEMA	NEGATIVE		32	
6.	CONGESTION, MILD EDEMA	HEMORRHAGIC MUCUS, SLIGHT		24	
7.	BRONCHOPNEUMONIA, CONGESTION, EDEMA	HEMORRHAGIC MUCUS, FOAM		28	
8.	EARLY BRONCHOPNEUMONIA	BLOOD TINGED MUCUS FOAM		25	
9.	CONGESTION, EDEMA	NEGATIVE		27	
10.	BILATERAL SEVERE BRONCHOPNEUMONIA	BLOOD TINGED YELLOW EXUDATE	NECROSIS, INFLAMMATORY EXUDATE	29	
11.	DIFFUSE BRONCHOPNEUMONIA	SUBMUCOSAL INJECTION, SLIGHT	POLYMORPHONUCLEARS IN LUMEN	30	
12.	EDEMA, STAPH. AUREUS BRONCHOPNEUMONIA	HEMORRHAGIC MUCOSA, PURULENT EXUDATE	NECROSIS, EDEMA, ULCERATION	41	
13.	EMBOLISM, PNEUMONITIS	TENACIOUS HEMORRHAGIC MUCUS		18	
14.	EMBOLISM, MASSIVE	LIQUID SECRETION IN BRONCHI		36	
15.	CONGESTION, EDEMA	THIN SANGUINOUS EXUDATE		10	
16.	BRONCHOPNEUMONIA, CONGESTION, EDEMA	HEMORRHAGIC YELLOW MUCUS, SLIGHT		35	
17.	EMBOLISM	INJECTED, SLIGHT SECRETION		37	
18.	BRONCHOPNEUMONIA, EMPHYSEMA, ATELECTASIS	NARROW LUMEN	FIBROUS THICKENING	29	



is reviewed, and the pertinent respiratory findings are charted on Fig. 1. Apparently in all the cases, except No. 18, where death was due to chronic tracheal and bronchial obstruction, and two others, where death was due to pulmonary embolism, death was due to circulatory collapse of central origin. All the cases listed had a tracheotomy, and they underwent numerous instances of blind suctioning with soft rubber catheters,

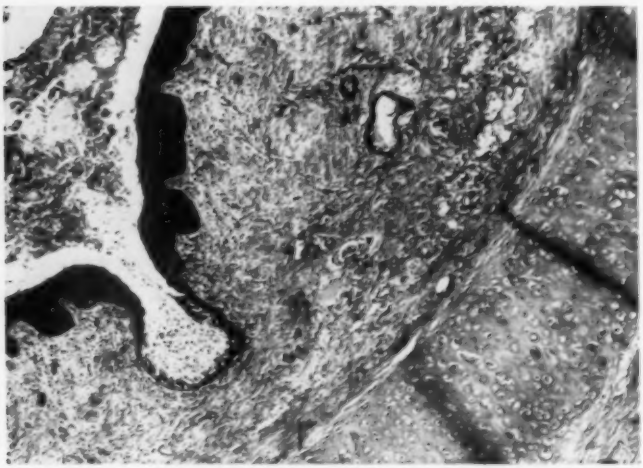


Fig. 2. Inferior extent of cicitricial scar in Case 18, showing debris in the lumen of the trachea, stratified squamous epithelium, and underlying scar tissue.

as well as bronchoscopy; however, it is believed that these procedures were quite frequent and that the incidence of suctioning procedures was roughly proportional to the time span involved. It is also assumed that the pathologic changes in the tracheobronchial tree are due principally to direct trauma, but they may be contributed to by pulmonary complications of respiratory paralysis or to pulmonary embolism where the latter has been shown to occur.

Case No. 18 on the above chart, which is of great interest in this study, is reviewed briefly:

N.K.E. (MGH No. 904807), a 29-year-old housewife, was admitted on September 5, 1955, with a diagnosis of questionable poliomyelitis, following a four day history of sore throat, which had also been present transiently in other members of the family, stiff neck of three days' duration with back pain and headache appearing shortly after the stiff neck. Her only significant physical findings were marked stiffness of the neck and bilateral limitation of straight leg raising. Her temperature was 101.0° F. Spinal fluid was under initial pressure of 114 mm. of water, and it contained 260 wbc/mm^3, 80 per cent of which were lymphocytes and 20 per cent polymorphonuclears. The next day she developed diplopia, and the following day weakness of voice, dyspnea, and weakness of the face and

Fig. 3. Central portion of cicitricial scar in Case 18, showing thick stratified squamous epithelium and underlying scar tissue which contains some inflammatory cells. Tracheal glandular tissue is also seen.

all extremities. She was then placed in a respirator. On September 9th she had complete quadriplegia. Tracheotomy was performed. On that day her temperature was 103° F.

During the next 15 days the patient had several episodes of cyanosis, necessitating repeated catheter suctioning, and a bronchoscopy, the latter for removal of dried mucous plugs. Fever persisted despite multiple antibiotic therapy. On September 20th, tidal volume was very low, 100 cc. Bronchoscopy revealed crusted blood narrowing all bronchi; it could not be detached without producing bleeding points. Tidal volume remained low, but it could be brought to 250 cc. at times by repeated bronchoscopy and positive and negative pressure breathing. Portable chest roentgenograms were negative. She became very disoriented at times. On September 24th it was felt that she was improving. Bronchoscopy revealed no narrowing, but a grayish mucosa was seen. During the next month her course remained intermittently febrile. This was thought due to Staphylococcus aureus, alpha hemolytic Streptococcus,

and Hemophylus influenzae infection, these organisms having been cultured from material aspirated by bronchoscopy.

The patient also underwent several episodes of hypotension and sinus tachycardia. Blind aspirations and bronchoscopies were still frequent. On November 7th, on bronchoscopy it was noted that two inches below the tracheotomy there was a concentric narrowing, where the mucosa was injected and edematous, and it bled easily. On November 19th it was stated that she had been doing well. Although paralyzed completely below the neck, she was placed for a few minutes each day on a rocking bed. On November 22nd she developed cyanosis, which required very frequent suctioning and bag breathing. It was stated that the bronchi would not admit a catheter. Tidal volume was kept at 250 cc.; however, she became unresponsive and died ten hours later.

The post mortem report, in part, is as follows:

The tracheostomy opening is located in the upper end of the trachea and measures 1 cm. in diameter. The opening is situated above the thyroid isthmus. The proximal 5 cm. below the tracheotomy orifice is unremarkable. Beginning 5 cm. below the tracheotomy orifice an almost abrupt narrowing of the lumen is noted. This narrowing extends for a distance of 4.5 cm. down to the carina. The narrowed portion measures 0.8 cm. in circumference at the narrowest point. The wall of the trachea in this region is thickened and fibrotic, with a purplish red discoloration. The mucosal surface of the narrowed portion is covered by a considerable amount of fibrinous exudate, while the lumen is almost completely occluded by a mucoid tenacious and sometimes purulent material. The bifurcation is also slightly narrowed for a distance of 1.5 cm. on each side, gradually merging into a normal bronchus on each side. The wall is similarly thickened, and the mucosal surface is covered by a fibrinous exudate. Beyond 1.5 cm. from the bifurcation the lumen appears normal; however, all the ramifications of the bronchial tree in either side reveal a large amount of mucoid and fibrinous exudate within the lumen, almost completely filling these structures. This exudate extends into the bronchi and bronchioles and can be expressed easily at the terminal portions of the lungs.

Microscopically, there is thickening of the mucosa by dense fibrous tissue infiltrated by chronic inflammatory cells. Squamous metaplasia of the epithelium and mild vascularization of the tunica propria are present. The tracheal glands show periglandular inflammatory reaction and dilatation of the ducts, or even of the glands themselves, and are filled by mucinous material. Foci of ulceration covered by fibrin are noted on the epithelial surface. The mucosal thickening is worse in the distal than in the proximal trachea. The fibrosis sometimes extends beyond the cartilage rings. The lumen contains cellular debris, mucus, inflammatory cells, red cells, and fibrin.

The prosector commented that this patient died of upper respiratory tract obstruction resulting from a complication, probably brought about by frequent bronchoscopic examination and chronic infection. The cicitricial contraction produced further difficuty in the aspiration of mucus, initiating a vicious cycle. Chronic irritation further down the bronchial tree has apparently produced bronchiolitis with subsequent organization of the exudate. This process was apparently

not diffuse, so that it could not have contributed significantly to the obstruction.

ANATOMIC DIAGNOSES.

Poliomyelitis, spinal.

Tracheitis and bronchitis, chronic, with tracheobronchial stenosis and obstruction.

Bronchiolitis fibrosa obliterans, focal.

Bronchopneumonia, mild.

Pulmonary atelectasis, superficial, bilateral.

Pulmonary emphysema, focal, mild.

Phlebothrombosis, old, right femoral vein.

Hydrothorax, left (50 ml.).

Cortical atrophy, adrenal, secondary to hydrocortisone therapy.

Focal necrosis of liver, old, with bile pigmentation of Kupffer Cells (Thorazine therapy).

Operation: Appendectomy, ten years.

Caesarean sections, three-and-a-half and two years.

Tracheostomy, two-and-a-half months.

Of those patients who survived severe poliomyelitis, it is not known that prolonged suction through a tracheotomy caused any permanent disability associated with injury to the tracheobronchial tree.

A six-year-old boy, a poliomyelitis patient, had a tracheotomy tube in place for 114 days. During the five months after closure of the tracheotomy wound, he attained, gradually, a nearly normal existence for a person of his age, including participation in competitive games; however, he died at home very suddenly, presumably because of aspiration of a pea, due to weak throat muscles. Unfortunately, a post mortem examination was not performed.

DISCUSSION.

It should be emphasized that the last case on the chart is the only one which showed scar tissue grossly.

It is also noteworthy that in several cases at necropsy, little material was found in the air passages, this fact attesting to the effectiveness of suctioning procedures. Under direct visualization, through tracheal windows, in five fresh cadavers, catheter suctioning was done under the usual pressures. The ability of the catheter to remove all visible material in the vicinity was striking, and it was noted also that the mucosa of the air passages was not drawn into the holes of the catheter.

SUMMARY AND CONCLUSIONS.

1. Suctioning procedures in the tracheobronchial tree cause acute and chronic inflammatory reactions in the trachea and primary bronchi, as seen in a series of 16 of 18 fatal poliomyelitis cases.

2. The degree of traumatic effect of suctioning does not seem to be clearly directly proportional to the length of time over which frequently performed suctioning is carried out.

3. One fatality in this series can be attributed to the mechanical effect of frequent suctioning over a very long period of time, a cicatricial tracheal scar.

4. In rehabiliated poliomyelitis cases, there is no commonly known morbidity due to prolonged suctioning of the tracheobronchial tree.

REFERENCES.

CARDON, J.: Tracheobronchial Aspiration with a Urethral Catheter. *Jour. A.M.A.*, 142:1039-1044, 1950.

HAIGHT, C.: Intratracheal Suction in the Management of Postoperative Pulmonary Complications. *Ann. of Surg.*, 107:218-228, 1938.

HAIGHT, H., and RANSOM, H. F.: Observations on the Prevention and Treatment of Postoperative Atelectasis and Bronchopneumonia. *Ann. of Surg.*, 114:243-262, 1941.

JACKSON, C., and JACKSON, C. L.: "Bronchoesophagology." W. B. Saunders Co., Philadelphia, 1950.

NELSON, T. G.; PEDICO, H. K., and BOWERS, W. F.: Use of Tracheotomy in Association with Artificial and Controlled Respiration. *Anesthesiol.*, 18:77-87, Jan.-Feb., 1957.

WEYL, R.: Tracheobronchial Aspiration as a Bedside Procedure, Indications and Limitations. *Amer. Jour. of Med. Sci.*, 222:66-72, 1951.

ZUCK, D.: Technic of Tracheo-Bronchial Toilet in the Conscious Patient. *Anaesthesia*, 6:226-230, 1951.

Gardner State Hospital.

THE GOULD AWARD.

The International Committee for the Gould Award announces the selection of the recipient for the year 1959—Professor Joel J. Pressman, M.D., University of California at Los Angeles, for his anatomic and radio-isotope studies of laryngeal compartments.

The Gould Award is presented annually for basic research in laryngology to encourage and reward fundamental investigations in this field. The International Committee for the Gould Award for the year 1960 includes: Sir Victor Negus, London, England; Professor Cotoji Satta, M.D., Tokyo, Japan; Professor Dr. Luzius Ruedi, Zurich, Switzerland; and Hans von Leden, M.D., Medical Director, The William and Harriet Gould Foundation, 30 North Michigan Avenue, Chicago 2, Ill., U. S. A.

The Gould Award consists of a plaque and an honorarium of $300.00.

TEMPOROGRAM.*

LESTER A. BROWN, M.D.,
and
DAVID T. SMILEY, M.D.,
(By Invitation),
Atlanta, Ga.

Tympanotubal lavage is used in the conservative treatment of middle ear infections especially when the tympanic membrane perforation is central. Medications used include broad spectrum antibiotics, steroid preparations and other medicaments in powder, liquid and semi-solid states.

In order to determine to what extent the medication was reaching the middle ear and connecting areas, several patients were studied radiographically after using a radiopaque dye for tympanotubal lavage.

SELECTION OF CASES.

The patients selected had perforations of the tympanic membranes; but otherwise were selected at random with two exceptions: *a.* where there had been no aural discharge for a long period of time, and *b.* young children whose lack of cooperation prevented obtaining satisfactory X-ray films.

TECHNIQUE.

The external auditory canal is carefully cleansed. The patient is instructed to lean his head toward the shoulder opposite the involved side, and to incline the body likewise until the vertical axis of the head is nearly horizontal. The face is turned upward and the head tilted backward. The upper external auditory canal is half filled with Lipiodol® (iodized oil—28 per cent iodine, Andre Guerbet and Company),

*Read at the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc., New Orleans, La., Jan. 15, 1960.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Jan. 22, 1960.

or aqueous Dionosil® (50 per cent propyliodone, n-propyl ester of 3:5 diiodo-4-pyridone-n-acetic acid, Glaxco Laboratories Ltd.). Negative pressure in the tympanum is created by drawing the air from it with a Bruening Zeiss pneumatic otoscope, the speculum of which fits air tight into the external auditory canal. Air drawn from the tympanum bubbles through the fluid in the external auditory canal. The procedure is continued until bubbles cease to appear. The fluid in the external auditory canal runs into the middle ear because

Fig. 1.

of the negative pressure. A cotton plug is inserted into the canal. Roentgenography is carried out immediately, in the positions commonly used for temporal bone study. These include the Law position with the tube tilted 15 and 35 degrees, the submento-vertex and the Towne positions, the last named made in stereo.

STATISTICS.

Ten patients were studied. The ages ranged from five to 68 years. Three persons were white, seven colored; six were male, four female. Some middle ears were free of discharge at the time of examination, whereas others revealed varying degrees of mucosal disease and ossicular destruction. Two

ears contained cholesteatoma, clinically. Two perforations were of the attic type, the others central. The quantity of dye required in each case was proportional to the extent of mastoid pneumatization and averaged 1.6 cc. The time required for satisfactory instillation ranged from two minutes to eight minutes. There was no difference between the oily (Lipiodol) and aqueous (Dionosil) contrast media in ease of instillation or ability to fill the air spaces, if the Dionosil was diluted by adding 3 cc. of water to the 20 cc. vial. Table I shows the distribution of the dye.

TABLE I.

Type of Perforation	No. of Cases	Dye Present in: Tympanum	Epitympanum	Eustach. Tube	Antrum (Mastoid)	Cells
Central	8	8	7	6	6	4*
Attic	2	2	2	1	2	1†

*Four others were acellular.
†One other was acellular.

COMMENT.

Scattered incomplete mastoid filling occurred in the first case, presumably because of insufficient time spent in the procedure, and an unusually long delay in obtaining the X-ray films; however, when the procedure was correctly performed, there was fairly complete filling of the mastoid cells with the dye, whether the cells were many or few.

In each case where the perforation involved Shrapnell's membrane, and there was cholesteatoma present, the mastoid process filled poorly.

The purpose of the study originally was to determine to what extent the medication reached the diseased areas. It becomes obvious that there are ramifications. For example, this procedure may be valuable in determining to what extent granulation or other soft tissue fills the aerated cavities of the temporal bone.

It would seem that the factors influencing the effectiveness of tympanotubal lavage include:

1. The size of the perforation.

2. The location of the perforation.

3. The presence and extent of cholesteatoma or granulation tissue.

4. The time spent in performing the procedure.

5. The position of the patient's head during the instillation.

Fig. 2.

No indication of residual dye was seen in the roentgenograms of three patients, which were repeated ten days after the original instillation.

REVIEW OF LITERATURE.

Dye contrast roentgenography of the air spaces of the temporal bone is not brand new. In 1928 Ruttin[1] was able to demonstrate the attic and mastoid antrum using 40 per cent *Iodopin* as contrast media. The following year, in this country, Mandelbaum[2] published his findings. He had been using *Lipiodol* for local treatment of chronic middle ear disease and found unexpectedly that the dye was useful as a diagnostic aid in outlining soft tissue masses. Unger,[3] in New York, em-

ployed the technique of tympanotubal lavage to differentiate between pneumatic and cancellous bone, noting that the dye filled the former but not the latter. He used living subjects and cadaver specimens. In 1951 Faccini[4] in Italy, demonstrated roentgenographically the normal anatomy of the middle ear and adnexa by instilling *Joduron B* through a Eustachian catheter and the auditory tube. Shortly thereafter, Papshitskii,[5] in Russia, demonstrated cholesteatoma by means of iodine, barium, and bismuth compounds injected through an attic cannula. Hungary was represented in an article by Bollobás[6] in which he reported filling of the middle ear system with contrast media.

SUMMARY.

1. It is possible to apply medication directly to the diseased areas of the aerated parts of the temporal bone through a perforation in the tympanic membrane.

2. It is possible to demonstrate the distribution of radiopaque dye by roentgenography.

3. The presence of filling defects may become diagnostically significant.

REFERENCES.

1. Ruttin, E.: Darstellung des Attik-Antrums in Röntgenbild durch Jodipinfüllung. *Acta Otolaryngol.*, 12:198-211, 1928.

2. Mandelbaum, M. J.: Diagnostic and Therapeutic Value of Iodized Oil in Chronic Purulent Otitis Media and Chronic Mastoiditis. Preliminary Report. The Laryngoscope, 39:156-170, March, 1929.

3. Unger, Max: Contrast Roentgenography of the Pneumatic Cells of the Temporal Bone. *Amer. Jour. Roentgenol.*, 54:384-388, Oct., 1945.

4. Faccini, M.: Visualizzazione con Mezzi di Contrasto Opachi della Tuba Auditiva e delle Cavitá dell'Orecchio Medio nell'uomo. *Radiol. Med., Tor.*, 37:11:908-916, Nov., 1951.

5. Papshitskii, Iu A.: Primenenie Kontrastnykyh Veshchestv pri Rentgenologicheskom Issledovanii Visochnykh Kostei. *Vest. Otorinolaryngol.*, 16:6:43-46, Nov.-Dec., 1954.

6. Bollobas, B.: Das Auffüllen des Mittelohrhöhlensystems mit Röntgen-Kontrastmitteln. *Fortsch. Röntgenstrahl.*, 83:3:323-329, Sept., 1955.

In Memoriam

FELIX ROBERT NAGER

1877 - 1959.

Felix Robert Nager was born on December 9, 1877, in Lucerne, Switzerland. His father, Dr. Gustav Nager, was a well-known Otolaryngologist, devoted to the welfare and rehabilitation of the hard of hearing. Felix R. Nager attended the public schools in that city and later went to the Universities of Basle, Geneva, Heidelberg and Zurich, where he was given his Medical Doctor degree in 1904.

He began his post-graduate training in Pathology, as an assistant to Professor P. Ernst in Zurich. Subsequently, he worked with Hofrat Professor Frederick Bezold in Munich and from 1904-1906 with Professor Frederick Siebenmann in Basle, the "founder" of Otolaryngology in Switzerland.

He began private practice in Zurich and in 1908 started a personally conducted clinic in the evenings for indigent patients. In 1915 he became Professor of Otolaryngology and in 1919 was appointed Director and Chairman of the newly created independent Ear, Nose and Throat Clinic and Ear, Nose and Throat Out Patient Department of the Zurich University School of Medicine. Until then, Ear, Nose and Throat Departments in Switzerland existed only in Basle, Berne and Lausanne. From 1940-1942 he was Dean of the Medical School, and in 1948 he was retired as Emeritus Professor.

Professor Nager made great and extensive contributions to the literature of otolaryngology. Almost 200 scientific papers, in several languages, bear evidence of his profound interest in every aspect of otolaryngology and in every problem that arose during his clinical activity. Many articles are concerned with clinical and anatomical pathology of the nose, paranasal sinuses, nasopharynx, throat, larynx, trachea, bronchial tree and the esophagus. His surgical interests were manifold; thus he became a pioneer and expert in the paranasal approach to intrasellar tumors of the hypophysis, and bronchoesophagology and laryngeal surgery were greatly developed. His principal interest, however, was the morbid anatomy of the temporal bone. Numerous are the monographic publications on bone diseases involving the otic capsule; on inflammatory disease, specific and non-specific, as they affect the ear; on

developmental anomalies of the ear with or without association with facial-cranial and skeletal malformations, and on etiologies of forms of deafness. Many of these studies have enduring significance.

Professor Nager founded a histopathological laboratory which now houses a unique collection of temporal bone pathology. Visitors from the entire world were welcomed here, and in this laboratory many scientists were introduced to the morbid anatomy of the ear. A large number of them made important contributions during their stay in Zurich or later as Directors or Departments in the United States and elsewhere. This international exchange of ideas and close team work on scientific problems of mutual interest started many wonderful friendships, created close bonds between Zurich and many centers of the Anglo-Saxon world and in its own way contributed much to the resumption of friendly cooperations after the disruptive effects of two world wars.

Following the tradition of his masters, Bezold and Siebenmann, Felix R. Nager created his own well-known school of otolaryngology which produced excellent chairmen of departments, teachers, scientists and numerous outstanding specialists throughout his country, and elsewhere.

At the time of the 25th anniversary of his clinic and also on his 70th birthday, pupils, colleagues, and friends throughout the otolaryngic world honored Professor Nager with a Festschrift of Scientific papers.

His early recognition of the important scientific activity in the New World promoted his journey to America in the mid-twenties and his return visit in 1937. He often said that these trips added much to the knowledge of the visitor. They gave him some of his dearest friends and brought him a sincere admiration for American otolaryngology.

He was elected to numerous honorary memberships. In the United States he was an Honorary Member of the American Academy of Ophthalmology and Otolaryngology (1925), of the American Otological Society, Inc. (1937), of the American Laryngological Society (1937) and of the Harvey Society of

New York (1925). In 1939 he delivered the Sir Felix Semon Lecture at the University of London. He became an Honorary member of the Otological and Laryngological sections of the Royal Society and of the Royal Society of Medicine. In 1949 he was elected an Honorary Fellow of the Royal College of Surgeons of England. He was one of the founders of the Collegium Oto-Rhino-Laryngologicum.

On April 15, 1948, Professor Nager retired as chairman of the department. After retirement, he continued a limited private practice, and devoted much time to the social and personal welfare of the hard of hearing and to his research. A progressive illness finally wrested his beloved activities from his hands.

His last address to the students and faculty was at the end of his 79th semester and had the title: "After Forty Years." In this address he cast a retrospective glance at the development of our specialty and he outlined the problems that will challenge otolaryngology in the future. In a philosophical manner he discussed dignity, virtues and ethics of the physician and reveals his personal philosophy or way of life to the coming medical generation from which he is departing. His valedictory address will always be remembered by those who were in the audience.

Professor Nager died at home on September 7, 1959, in his 82nd year. He is survived by his widow, the former Emily Reinhart, by three daughters and three sons.

ROYAL SOCIETY OF MEDICINE.

The Sections of Laryngology and Otology of the Royal Society of Medicine, London, will be meeting on May 6 and July 14-16, 1960. The programs follow:

May 6 Meeting.

Otology. 10 A.M.—Discussion on the Effects of Noise Upon Hearing.
Dr. A. Glorig, Los Angeles, Calif.

Laryngology. 2:30 P.M.—"Dysphagia in Adults (non-malignant), Its Diagnosis and Management."
Mr. H. D. Brown Kelly
"Management of Chemical Burns of the Oesophagus."
Professor Truls Leegaard (Oslo).
Mr. Norman Barrett will open the discussion.

July Meeting.

The Sections will hold a combined meeting at Oxford and the program follows:

15th, 9:30 A.M.—"The Innervation of the Inner Ear."
Professor Arslan (Padua)
Also papers by members of the Otological Research Unit, Medical Research Council, Queen Square, London.
2:30 P.M.—Visit to Department of Plastic Surgery, Churchill Hospital.

16th, 9:30 A.M.—The Management of Frontal Sinusitis and its Complications."
Mr. Desmond Dawes.
"The Radiography of the Lower Lateral Food Channels."
Dr. F. H. Kemp and Dr. G. M. Ardran.

Members of the American Laryngological Association, the American Triological Society and the American Otological Society who will be in England at these times will be welcome. If they wish to come they should write to: E. H. Miles Foxen, F.R.C.S., Secretary for Laryngology; Charles Gledhill, M.B.E., F.R.C.S., Secretary for Otology.

PROGRAM FOR AMERICAN MEDICAL ASSOCIATION SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY, 109th ANNUAL MEETING.

June 15-17, 1960, Miami Beach, Fla.

Wednesday, June 15, 1960.

Joint Session of the Section on Laryngology, Otology and Rhinology, Section on the Diseases of the Chest, and Section on Radiology.

1. Panel Discussion on Tumors of the Trachea and Bronchi.

   Moderator: Dr. Paul H. Holinger, Chicago 11, Ill.

   Endoscopist: Dr. Herbert W. Schmidt, Rochester, Minn.

   Radiologists: Dr. Barton R. Young, Philadelphia, Pa.; Dr. Wendell G. Scott, St. Louis, Mo.

   Surgeons: Dr. Robert J. Jensik, Chicago 4, Ill.; Dr. Robert W. Jamplis, Palo Alto, Calif.

2. Panel on Disseminated Diseases of the Chest.

   Moderator: Dr. Harold O. Peterson, Minneapolis, Minn.

   Internist: Dr. James A. Wier, Denver, Colo.

   Chest Physician: Dr. David Radner, Chicago, Ill.

   Endoscopist: Dr. Walter H. Maloney, Cleveland 6, O.

   Radiologists: Dr. Eugene F. Van Epps, Iowa City, Ia.; Dr. Clyde A. Stevenson, Spokane, Wash.

   Surgeons: Dr. Donald L. Paulson, Dallas, Tex.; Dr. William Tuttle, Detroit, Mich.

Thursday, June 16, 1960.

Audiology: Its Progress and Problems.

Kenneth O. Johnson, Ph.D., Executive Secretary, American Speech and Hearing Association, Washington 6, D. C., (By Invitation).

External Otitis: Specific Therapy Based on Accurate Diagnosis.

Edley H. Jones, Vicksburg, Miss.

Address of Guest of Honor.

Louis H. Clerf, St. Petersburg 2, Fla.

Nasal Tip Surgery.

Sam H. Sanders, Memphis 4, Tenn.

Address of Section Chairman.

Paul H. Holinger, Chicago 11, Ill.

Problems and Complications in Head and Neck Surgery.

George A. Sisson, Syracuse 10, N.Y., and Norman E. Johnson.

The Long-Term Care of the Laryngectomized Patient.

George F. Reed, Massachusetts Eye and Ear Infirmary, Boston, Mass.

Friday, June 17, 1960.

Cystadenoma of the Parotid and of the Larynx.

Arthur J. Kuhn, Hammond, Ind.

Effect of Premarin on the Ground Substance.

Maurice Schiff, Capt. (MC) USN, U. S. Naval Hospital, Oak Knoll, Calif.

Cervico-Facial Congenital Anomalies.

Richard T. Farrior, Farrior Clinic, Tampa 6, Fla.

Incidence of Hearing Impairment in a Medical Center Population.

Edmund P. Fowler, Jr., M.D., and Thomas H. Fay, Ph.D., New York 32, N. Y.

Otometric Operations.

George D. Albers, Grand Rapids 3, Mich.

Five Year Results of Stapes Mobilization and Current Results with Vein Plug Stapedeoplasty.

C. M. Kos, University Hospitals, Iowa City, Ia.

Fenestration of the Oval Window.

John J. Shea, Jr., Memphis 4, Tenn.

Use of Ear Canal Skin in Myringoplasty.

William F. House, Los Angeles 57, Calif.

Problems with Tympanoplasty.

Edward C. Brandow, Jr., Albany 10, N. Y.

PAN-PACIFIC SURGICAL ASSOCIATION.

Eighth Congress.

Honolulu, Hawaii, September 27-October 5, 1960.

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 27 through October 5 in 1960.

All members of the profession are eligible to register and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities because of limited space.

An outstanding scientific program by leading surgeons promises to be of interest to all doctors. Ten surgical specialty sections are to be held simultaneously.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1960.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

President: Dr. J. J. Longacre, 1503 Carew Tower, Cincinnati, O.
President-Elect: Dr. D. C. Samuel Pruzansky, D.D.S., 840 So. Wood St., Chicago, Ill.
Secretary-Treasurer: Dr. Spriestersbach, Ph.D., Department of Otolaryngology, University Hospital, Iowa City, Ia.
Meeting: Palace Hotel, Denver, Colo., May 12-14, 1960.

AMERICAN BOARD OF OTOLARYNGOLOGY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee Dr., Syracuse 10, N. Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.
Meeting: Palmer House, Chicago, Ill., October, 1960.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Verling K. Hart, 107 W. 7th St., Charlotte, N. C.
Vice-President: Dr. Daniel C. Baker, Jr., 903 Park Ave., New York, N. Y.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3, Pa.
Treasurer: Dr. Charles M. Norris, 3401 Broad St., Philadelphia, Pa.
Meeting: Deauville Hotel, Miami Beach, Fla., March 15-16, 1960 (Afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. W. J. McNally, Montreal, Canada.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.
Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Edwin N. Broyles, Baltimore, Md.
Meeting: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
President-Elect: Dr. Fletcher D. Woodward, 400 Locust Ave., Charlottesville, Va.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Treasurer: Dr. K. M. Day, 121 University Pl., Pittsburgh, Pa.
Annual Meeting: Deauville Hotel, Miami Beach, Fla., March 13-19, 1960.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Paul H. Holinger, Chicago, Ill.
Vice-Chairman: Dr. Lawrence R. Boies, Minneapolis, Minn.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, Ohio.
Section Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Dean M. Lierle, Iowa City, Ia.
Meeting: Miami Beach, Fla., June 13-17, 1960.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. R. C. Martin, 384 Post St., San Francisco 8, Calif.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14, Minn.
Meeting: Deauville Hotel, Miami Beach, Fla., March 13-14, 1960.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worcester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Roland M. Loring, 25 E. Washington St., Chicago, Ill.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland 17, Ore.
Annual Clinical Session: Chicago, Ill. (Definite time and place to be announced later).
Annual Meeting: October 1, 1960, Belmont Hotel, Chicago, Ill.

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY.

President: Dr. John J. Conley, New York, N. Y.
Vice-President: Dr. Joseph H. Ogura, St. Louis, Mo.
Treasurer: Dr. F. Johnson Putney, Philadelphia, Pa.
Secretary: Dr. George A. Sisson, Syracuse, N. Y.
Annual Meeting: Palmer House, Chicago, Ill., October 1, 1960.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Oscar J. Becker, Chicago, Ill.
Vice-President: Dr. Sam H. Sanders, Memphis, Tenn.
Treasurer: Dr. Joseph C. Miceli, Brooklyn, N. Y.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28, N. Y.
Meeting: Chicago, Ill., October, 1960.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Walter E. Owen, Peoria, Ill.
President-Elect: Dr. Leland H. Prewitt, Ottumwa, Ia.
Vice-President: Dr. D. A. Skinner, Newark, O.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting: Palmer House, Chicago, Ill., October 8, 1960.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Alberto Gallo.
First Secretary: Dr. Alfredo Martinelli.
Second Secretary: Dr. Guedes de Melo Neto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. Antonio de Almeida, Dr. Gabriel Pôrto, and Dr. Roberto Franco do Amaral.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perellò, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. Gordon H. Francis, 925 W. Georgia St., Vancouver, B. C.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Road, Halifax, Nova Scotia.
Meeting: Jasper Park Lodge, Jasper National Park, Alberta, June 10-12, 1960.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. E. Hartenbower, 203 N. Main St., Bloomington, Ill.
President-Elect: Dr. Edgar T. Blair, Springfield, Ill.
Vice-President: Dr. G. LeRoy Porter, Urbana, Ill.
Delegate at Large: Dr. S. G. Baldwin, Danville, Ill.
Secretary-Treasurer: Dr. C. L. Pannabecker, Peoria, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. George Woodruff, Woodruff Clinic, Joliet, Ill.
Vice-President: Dr. Linden Wallner, 122 So. Michigan, Chicago, Ill.
Secretary-Treasurer: Dr. Robert Lewy, 25 East Washington St., Chicago 2, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. John E. Arthur.
Secretary: Dr. M. L. Battles.
Meetings: First Monday of October through May, University Club, Columbus, O.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. James A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. Dekle Taylor, Jacksonville, Fla.
President-Elect: Dr. Kenneth S. Whitmer, Miami, Fla.
First Vice-President: Dr. William H. Anderson, Jr., Ocala, Fla.
Second Vice-President: Dr. Marion W. Hester, Lakeland, Fla.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr., 1 Davis Blvd., Tampa 6, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Mariano C. Caballero.
Vice-President: Dr. Joseph Freeman.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting: Quarterly in March, May, October and December on the second Thursday of the month, 6:30 P.M., at the McAllister Hotel, Miami, Fla.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Lucian W. Alexander, 921 Canal St., New Orleans, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., April 8-9, 1960.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairman in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Petrarca 332-1, Mexico 5, D. F.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY. (Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Ohrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. David D. DeWeese, 1216 S. W. Yamhill St., Portland 5, Ore.
Secretary-Treasurer: Dr. Paul B. Myers, 223 Medical Dental Bldg., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Henry Thiele Restaurant, 23rd and W. Burnside, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. E. H. Campbell, 133 So. 36th St., Philadelphia 4, Pa.
Secretary-Treasurer: Dr. Raymond Jordan, 121 University Place, Pittsburgh, Pa.
Meeting: Palmer House, Chicago, Ill., October, 1960.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. John F. Tolan, 1118-9th Ave., Seattle 5, Wash.
Secretary-Treasurer: Dr. Homer E. Smith, 686 Twelfth Ave., Salt Lake City, Utah.
Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 No. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. John J. O'Keefe.
Vice-President: Dr. Joseph P. Atkins.
Secretary: Dr. William A. Lell.
Executive Committee: Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, Dr. William A. Lell, Dr. William J. Hitschler, and Dr. Chevalier L. Jackson.

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Cesar F. Villafuerte.
Vice-President: Dr. Napoleon C. Ejercito.
Secretary-Treasurer: Dr. Eusebio E. Llamas.
Directors: Dr. Antonio L. Roxas and Dr. Armando T. Chiong.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Vice-President: Dr. Carson S. Demling, 513 Jenkins Bldg., Pittsburgh 22, Pa.
Secretary-Treasurer: Dr. Clyde B. Lamp, 8101 Jenkins Arcade, Pittsburgh 22, Pa.

PORTUGUESE OTORHINOLARYNGOLOGICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

SIXTH INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST.

Meeting: University of Vienna, August 29 to September 1, 1960.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Morris E. Krucoff.
Vice-Chairman: Dr. Max J. Fischer.
Secretary: Dr. Adrian J. Delaney.
Treasurer: Dr. Robert D. Ralph.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: Dr. F. T. Land, 13 Newton Place, Glasgow, C. 3.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow, C. 3.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA
Monterrey 47-201
Mexico 7, D. F.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela.
Treasurer: Dr. Benito Madariaga.
First Vocal: Dr. Rafael González.
Second Vocal: Dr. Juan Oberhauser.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael Garcia Tapia.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DEL NORESTE.

Presidente: Dr. Livio M. Lataza C.
Secretario: Dr. Ramón Prieto.
Tesorero: Dr. José Gomez Galizia.
Vocales: Dres. Enrique del Buono y O. Benjamin Serrano.

SOCIEDAD VENEZOLANA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Gabriel Briceño Romero.
Vice-Presidente: Dr. Silvestre Rincón Fuenmayor.
Secretario General: Dr. Oscar Bustamante Miranda.
Tesorero: Dr. Arturo Marrero Gómez.
Vocales: Dr. Miguel Octavio Russa, Dr. Benjamin Briceño, Dr. Oscar Gonzalez Castillo.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DO RIO GRANDE DO SUL.

President: Dr. Ivo Adolpho Kuhl.
Secretary: Dr. Decio Lisboa Castro.
Treasurer: Dr. Jorge Valentin.

SOCIEDAD PANAMENA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Manuel Preciado.
First Vice-Presidente: Dr. Alonso Roy.
Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. María Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Lt. Col. Stanley H. Bear, USAF (MC), USAF Hospital, Maxwell (Air University), Maxwell Air Force Base, Ala.
Secretary-Treasurer: Capt. Maurice Schiff, MC, USN, U. S. Naval Hospital, Oakland, Calif.
Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. L. D. Lide, Florence, S. C.
President-Elect: Dr. Harry Ross, Anderson, S. C.
Vice-President: Dr. David Stack, Spartanburg, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. C.
Meeting: Jointly with the North Carolina Eye, Ear, Nose and Throat Society in Greenboro, N. C. Headquarters will be in the King Cotton Hotel, Sept. 13-15, 1960.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. George M. Haik, Professor and Head of the Department of Ophthalmology at Louisiana State University School of Medicine, 812 Maison Blanche Building, New Orleans 16, La.
Chairman-Elect: Dr. Mercer G. Lynch, Assistant Professor of Otolaryngology, Tulane University School of Medicine, 3503 Prytania St., New Orleans 15, La.
Vice-Chairman: Dr. Bernard J. McMahon, Director of the Department and Clinical Professor of Otolaryngology, St. Louis University School of Medicine, 8230 Forsythe Blvd., Clayton 24, Mo.
Secretary: Dr. Albert C. Esposito, First Huntington National Bank Building, Huntington, W. Va. Formerly instructor of Ophthalmology, Ohio State University College of Medicine, Columbus, O.
Meeting: St. Louis, Mo., October 31, 1960, to November 3, 1960.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Va.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Va.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Nime K. Joseph, Wheeling, W. Va.
President-Elect: Dr. John A. B. Holt, Charleston, W. Va.
Vice-President: Dr. William K. Marple, Huntington, W. Va.
Secretary-Treasurer: Dr. Albert C. Esposito, Huntington, W. Va.
Director for Two Years: Dr. James T. Spencer, Charleston, W. Va.

INTERNATIONAL POSTGRADUATE COURSE IN RECONSTRUCTIVE SURGERY OF THE EXTERNAL NASAL PYRAMID AND NASAL SEPTUM.

An international course in Reconstructive Surgery of the External Nasal Pyramid and Nasal Septum will be given in Mexico City July 4-15, 1960, under the auspices of the University of Mexico Medical School and its Postgraduate Department, in cooperation with the American Rhinologic Society, and under the direction of Dr. Andres Bustamante Gurria, of Mexico City. The guest professor will be Dr. Maurice H. Cottle, Chicago Medical School. He will be assisted by a faculty of otorhinolaryngologists from the United States and Mexico.

Class membership is limited. Apply for registration information to Dr. Robert M. Hansen, Secretary, American Rhinologic Society, 1735 North Wheeler Ave., Portland 17, Ore.

NOTICE TO CONTRIBUTORS

THE LARYNGOSCOPE reserves the right of exclusive publication of all articles submitted. This does not preclude their publication in Transactions of various Societies.

Manuscripts should be typewritten, double spaced, on one side of paper only and with sufficient margins to allow for corrections.

Author's name and city should appear directly under title on first page; street address at end of article.

All prints or photographs to be submitted in black and white, in good sharp contrast. Good halftones depend upon clear photographs. Line drawings for zincs to be in black and white. Colored inks or red or blue quadrille rulings will not reproduce.

References should be complete: author's surname, initials, title of article, Journal, volume, page, month, year.

Six illustrations will be furnished for each article without cost to author. Authors will please limit illustrations to six or assume the expense of additional illustrations.

Proofs will be submitted to authors for corrections. If these are not returned, articles will be published as corrected in this office.

Reprints will be furnished at the following prices:

WITHOUT COVER

	250 Copies	500 Copies	1000 Copies	2000 Copies
Four Pages	$ 19.25	$ 23.00	$ 30.75	$ 44.50
Eight Pages	33.50	42.75	58.50	83.00
Twelve Pages	47.00	60.75	86.25	131.50
Sixteen Pages	61.00	78.75	98.75	146.75
Twenty Pages	76.00	96.25	129.50	187.25
Twenty-four Pages	88.75	112.50	150.00	217.25
Twenty-eight Pages	97.50	123.25	162.25	233.50
Thirty-two Pages	115.00	139.75	180.00	267.00

WITH COVER

Four Pages	$ 37.25	$ 46.50	$ 61.50	$ 88.75
Eight Pages	51.50	66.25	89.25	127.25
Twelve Pages	65.00	84.25	117.00	175.75
Sixteen Pages	79.00	102.25	129.50	191.00
Twenty Pages	94.00	119.75	160.25	231.50
Twenty-four Pages	106.75	136.00	180.75	261.50
Twenty-eight Pages	115.50	146.75	193.00	277.75
Thirty-two Pages	133.00	163.75	210.75	311.25

Express charges will be paid by consignee.